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Lack of Relationship Between Vehicle Damage and Occupant Injury

Malcolm C. Robbins
Robbins & Assocs.

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Lack of Relationship Between Vehicle Damage and Occupant Injury

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ABSTRACT

A common misconception formulated is that the amount of vehicle crash damage due to a collision, offers a direct correlation to the degree of occupant injury. This paper explores this concept and explains why it is false reasoning. Explanations with supporting data are set forth to show how minor vehicle damage can relate or even be the major contributing factor to occupant injury. Mathematical equations and models also support these findings.

INTRODUCTION

A common concept formulated is that the amount of motor vehicle crash damage offers a direct correlation to the degree of occupant injury. This paper explores this concept and explains why it is false reasoning. This false reasoning is often applied by insurance adjusters, attorneys and physicians and frequently results in costly unjustified litigation. Due to this litigation process, the injured parties often are not compensated, resulting in unjustified hardship to the party who has already been injured.

The object of this paper is to present a clear understanding of vehicle body performance when it is subjected to crash dynamics and the relationship to occupant dynamic responses and resulting injury.

THEORY

One of the major factors relating to occupant injury due to a collision is the G force to which the occupant is subjected. [1][2] Even with seat belts air bags and other measures, severe injury and fatality occurs when a vehicle is subject to a collision. [3][4][5][6] This is a rather complex subject to answer in a single paper, but fundamentally even when seat belts are used, the G force sustained by the vehicle beyond the crush zone or arresting distance is transferred to the occupant.

Galileo Galilei formulated an equation that can be used to demonstrate the G force an occupant will receive, assuming a "fixed" seated position. If an object starts from rest, Galilei's equation states: [1]

$$V = \sqrt{2as} \qquad (1)$$

where V = Velocity of object
a = acceleration rate
s = distance moved by object.

Rearranging Equation 1 to get deceleration, we have:

$$a = \frac{V^2}{2s} \qquad (2)$$

where s = arresting or crush distance
V = Velocity at time of impact
a = deceleration

Applying this formula (2) to the scenario of a pole vaulter. If a pole vaulter jumps 6.5 meters (20 feet), his speed when reaching a 1.5-meter (5-foot) safety mat can be calculated thus, using Equation 1:

$$V = \sqrt{2as} \qquad (1)$$

where $s = 6.5 - 1.5 = 5$ meters
$a = 9.81$ m/sec^2

hence: V = 11.29 m/sec or 40 km/hr (25 mph)

The resulting G force to which the pole vaulter is exposed can be calculated to be as follows, using Equation 2;

$$a = \frac{V^2}{2s} \qquad (2)$$

where V = 11.29 m/sec
$s = 1.5 - 0.5 = 1$ meter

hence: a = 63.7 m/ sec^2 (6.5 Gs)

If the vaulter impacted a concrete surface, the results would be clearly different. It is the amount of crush in the safety padding that prevents injury to the pole vaulter.

Applying the formula to vehicles which impact a solid brick wall:

First Scenario: Vehicle Green is traveling at a velocity of 12 meters/second (25 mph) and crushes 1 meter (3.1 feet) while impacting a solid brick wall. Using equation (2) above, then V = 8 m/sec (25 mph), s = 1 m (3.1 feet) and deceleration is:

$$a = \frac{12^2}{2} = 72m/sec^2 = 7G$$

Second Scenario: Vehicle Red undertakes same velocity as Vehicle Green but crushes only 0.2 meters while impacting the solid brick wall. Deceleration is:

$$a = \frac{12^2}{0.2} = 360m/sec^2 = 37G$$

The results show that the greater the crush distance of the vehicle, the less the G force received by the occupant.

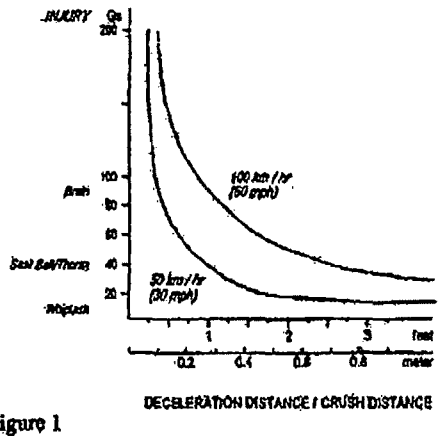

Figure 1

The graph shown in Figure 1 demonstrates the effect a vehicle's crushing distance has on the G force with a fixed collision speed.

Discussion

The average force an occupant of a motor vehicle experiences with normal driving is in the range of 0.2 to 0.5 G. Under these conditions motor vehicle occupants can readily change direction or speed with the vehicle. Some braking will impose a resulting force of 0.9 G, which may cause unrestrained occupants to be thrown forward in the occupant area. The limiting factor is the amount of coefficient of friction available between tire and road for braking and steering. Injury has been known to result due to severe braking, typically when occupants did not have time to brace themselves and were restrained.

If a motor vehicle impacts an object, loads on occupants can rise to very high values. When this takes place, the unrestrained occupant cannot keep pace with the vehicle's change in speed or direction. Hence the unrestrained occupant continues to move within the interior passenger compartment, colliding with the compartment surfaces such as steering wheel, windshield or dashboard. The introduction of seat belts is an attempt to keep the occupant restrained and moving along with vehicle speed and direction. Air bags and padded interior surfaces are provided to cushion the occupant's limbs and head, which contact with occupant interior surfaces.

While the amount of crush a vehicle sustains does not relate to occupant injury, provided no penetration occurs to the occupant compartment, the amount of crush does relate to the impact velocity or speed in the event of a collision with another vehicle or object. In fact, evaluation of occupant injury when related to vehicle damage can only be made when several factors are taken into account. Some of these factors are the following:

Dynamics of force applied to occupant.

- Velocity of vehicle or objects on impact.
- Crushing or arresting distance of vehicle or object. [7][8]
- Ability of vehicle or objects to dissipate the energy of the impact.
- Combination of above factors will establish the dynamics of force applied to occupant.
- Initial positioning of occupant in relation to safety devices such as seat belts or air bags.

Physical condition of occupant.

- Degree of muscular stimulation at the time of impact, i.e., was impact anticipated by occupant?
- Structural strength of occupant, i.e., sex, age, bone mineral content and joint strength. [9]
- Geometric dimensions, i.e., height, weight.

One main factor for determining the dynamics of occupant injury due to a motor vehicle collision is the amount of crush or arresting distance, known as value "s" and previously discussed. This value can vary a great deal from vehicle to vehicle and its location on the vehicle. If we examine a soft drink extruded aluminum can and liken it to a motor vehicle body, several observations can be made:

- Firstly, force applied on the top of the can downwards meets a greater resistance than a force applied to the sides. Clearly the type of structure of the can plays a major part in the deformation resistance.
- Secondly, if a force is applied to the top, a relative great deal of resistance is initially met, then slowly, as the can is crushed, the amount of resistance deteriorates and the can yields.

Likewise, on a vehicle with a chassis, no serious visual deformation may occur even though it is subjected to relatively high speeds of impact. Classically, we see this in the case of pickup trucks or all-terrain vehicles that are traditionally fitted with a solid bumper-to-bumper chassis. Many of these types of vehicles are subjected to relatively severe impacts with little or no resulting damage to their bodies and bumpers. The classic whiplash injury associated with a great deal of litigation is most likely founded on the reasoning that if there was little or no vehicle damage, no injury can result. Motor vehicle bodies or bumper-to-bumper chassis offer little or no crushing effect on arresting obstacles when impacted; thus, relatively high G forces can be experienced by occupants when rear-ended, resulting in whiplash injury. The use of stiff motor vehicle bodies and chassis will also produce a spiked G force loading to occupants, even if little damage occurs to vehicle body or chassis.

Spike loading is a result of a non-linear yielding of a vehicle body, as previously discussed in the scenario of the tin

can. In actual practice, deceleration rates during an automobile collision are rarely uniform, especially when chassis, drive trains and mounting panels are involved in the collision.

It is not uncommon to see a motor vehicle that has experienced mass destruction and damage, yet the occupants sustained little or no injuries. This is often a prime example of a situation in which the vehicle or vehicles have absorbed the dissipating kinetic energy of the collision. The occupants are thus not subjected to severe G forces. It is for this very reason that racing cars, when seen in a collision, appear to almost shed their body structure. Wheels are seen detaching and the body structure is seen to dissipate and crush almost in every direction. High-performance racing cars as seen on the Grand Prix circuit are designed with state-of-the-art crash engineering. The main outside structure of these racing cars is designed to allow for crushing and to dissipate energy in the event of a collision. The driver is protected by a rigid enclosure and is also very effectively restrained. These design factors in high-performance crash engineering account for the low driver-injury rates, even though the collisions involve very high speeds. So here we see heavy vehicle-body damage and relatively low occupant injury rates. I.e., the body of the racing car is sacrificed to prevent driver injury or death.

Summary

The amount of crush or damage received by a motor vehicle in a collision is an indication of velocities involved when the stiffness of the motor vehicle and object or objects is known. However, the crush damage does not relate to the expected occupant injury, i.e., the more vehicle damage, the more chance that the occupant is injured, is not a conclusion that can be made. In fact, it is more likely the reverse. If the occupant is decelerated over a greater time/distance due to a large crush/arresting distance, then the likelihood of injury is reduced.

This conclusion has been demonstrated by both mathematical expression and practical examples. The first example is that of the pole vaulter who survives his 5-meter (16-foot) drop by the crush of the padding or mat. It is this crush which breaks the vaulter's fall and hence allows for increased stopping distance and time. The second practical example is that of the high-performance racing car which makes use of a rigid driver compartment for protection. However, the compartment is surrounded by a body which is designed to allow for crush or deformation due to a collision. The result is a reduced number of injuries or fatalities.

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About the Author

Malcolm C. Robbin's training and education began with a formal engineering apprenticeship in the UK. His qualifications include City and Guilds in Manufacturing and Mechanical Engineering and a B.Sc. degree in Production Engineering. He is a member of the American Society of Safety Engineers and the Institution of Production Engineers and Industrial Managers in the UK.

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Rear-end impacts: vehicle and occupant response.

Davis CG.

Abstract

BACKGROUND: There is a controversy regarding the likelihood of injuries sustained when one car strikes another at a relatively low speed with little or no vehicle damage. Plaintiffs often claim injuries whereas defendants counterclaim that injuries could not have occurred with such a relatively minor impact.

OBJECTIVE: To review the dynamics of low-speed rear-end collisions resulting in little or no visible damage and to decide whether occupant injury can occur; also, to discuss diagnostic examination and treatment that may be helpful to the clinical practitioner.

DATA SELECTION: A Medline search for articles discussing low-speed rear-end collisions was conducted. Other articles and studies were reviewed that discussed low-speed rear-end collisions and factors impacting the neuromusculoskeletal system relevant to clinical practitioners. Articles included were human low-speed rear-end tests, lab tests on cadavers, automotive engineering articles, and peer-reviewed journal articles on whiplash. A few live animal and simulation studies were considered for the background of possible injury mechanism and vehicular deformation. Excluded were non-rear-end collison and single case reports.

DATA SYNTHESIS: The data were studied to find a relationship between the resultant vehicle dynamics and occupant movement, biological mechanisms of injury and the neurological mechanisms causing complaints. Data were also studied to investigate objective findings supporting subjective complaints.

CONCLUSION: In low-impact collisions, there are usually no skid marks and minor or no visible damage to the vehicle. There is a lack of relationship between occupant injury, vehicle speed and/or damage. There does not seem to be an absolute speed or amount of damage a vehicle sustains for a person to experience injury. Crash tests indicate that a change of vehicle velocity of 4 km/hr (2.5 mph) may produce occupant symptoms. Vehicle damage may not occur until 14-15 km/hr (8.7 mph). Occupant soft tissue and joint injuries resulting from low-speed vehicle collisions respond positively to afferent stimulation of mechanoreceptors. The diagnosis of the occupant injuries relies on standard orthopedic neurological testing, autonomic concomitant signs and qualitative and quantitative testing.

Comment in

Rear-end impacts: vehicle and occupant response. [J Manipulative Physiol Ther. 2000]

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SURFACE VEHICLE INFORMATION REPORT

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Human Tolerance to Impact Conditions as Related to Motor Vehicle Design

RATIONALE

The members of the SAE Human Biomechanics and Simulations Standards Steering Committee have reviewed J885 and made a conscientious decision to stabilize this Information Report. Extensive research in human tolerance has been conducted since J885 was last revised. Several published papers present Injury Assessment Reference Values (IARVs) that are based on the current human tolerance data. The U.S. auto industry uses these values to assess the restraint performance of their vehicle designs. The committee members see no value in writing a SAE document to summarize these IARVs since their bases are well documented in the literature. J885 has historical value since many of the early IARVs were established from the data it contains.

STABILIZED NOTICE

This document has been declared "Stabilized" by the SAE Human Biomechanics and Simulations Standards Steering Committee and will no longer be subjected to periodic reviews for currency. Users are responsible for verifying references and continued suitability of technical requirements. Newer technology may exist.

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Foreword—This Document has not changed other than to put it into the new SAE Technical Standards Board Format.

TABLE OF CONTENTS

1. ***Scope—***This report reviews current[1] quantitative data on human tolerance levels without recommending specific limits. Data developed on humans (including cadavers) are presented where available; however, in many cases animal data are provided where no suitable human results have been reported. This report confines itself, as much as possible, to information of direct use to the automotive designer and tester. Data of only academic interest are largely omitted; therefore, J885 should not be considered as a complete summary of all available biomechanical data.

Most of the data cited in this report applies to adult males since little information is available on women or children. The summary data provided in the tables should be considered in conjunction with the accompanying descriptive test. This material explains the manner in which the data were obtained and provides an insight as to their limitations.

1.1 **Purpose—**The purpose of this report is to assist the automotive safety designer and tester by providing them with quantitative data on the strength of the human body under impact loading conditions.

2. ***References***

2.1 **Applicable Publications—**The following publications form a part of the specification to the extent specified herein.

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FMVSS 208

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3. Definitions

3.1 Abbreviated Injury Scale (AIS)—A numerical rating system for quantifying the severity of injuries to an accident victim. The rating scale is:

Code	Category
1	Minor
2	Moderate
3	Serious
4	Severe
5	Critical (survival uncertain)
6	Maximum (virtually unsurvivable)
9	Unknown

For further details see the Abbreviated Injury Scale 1980 Revision published by the American Association for Automotive Medicine.

3.2 Anterior—Front.

3.3 Anterior-Posterior (a-p)—Front to back; in humans, directed from the belly surface towards the back surface.

3.4 Articular—Pertaining to a joint.

3.5 Avulsion—Tearing away of a part.

3.6 Cancellous Bone—The spongy or lattice-like structure of a bone occurring towards its inner core.

3.7 Cartilage—Fibrous connective tissue.

3.8 Cervical—Pertaining to the neck.

3.9 Comminuted—Broken into small pieces.

3.10 Compact Bone—The dense structure of the bone which constitutes its outer portion.

3.11 Condyle—A rounded projection on a bone usually associated with a joint.

3.12 Contusion—Bruising from a direct impact.

3.13 Cricoid Cartilage—The ring shape fibrous tissue which encircles the airway passage near the top of the neck.

3.14 Distal—Remote; further away from the point of reference.

3.15 Esophagus—The passageway which carries food to the stomach.

3.16 Extension—Rearward bending when applied to the neck.

3.17 Femur—Thigh bone.

3.18 Fibula—The outer and smaller of the two bones of the lower leg.

3.19 Flexion—Bending; forward bending when applied to the neck.

3.20 Frontal Bone—The bone constituting the forehead and upper forward portion of the skull.

3.21 Functional Injury—A trauma which is not readily observable on visual examination but manifests itself as an impairment of normal usage or behavior.

3.22 Hemorrhage—Bleeding.

3.23 Hemothorax—A collection of blood in the sac surrounding the lungs.

3.24 Hyperextension—Extreme or excessive extension of a limb or part; backward overbending when applied to the neck.

3.25 Hyperflexion—Extreme or excessive flexion of a limb or part; forward overbending when applied to the neck.

3.26 Inferior—Below.

3.27 Inferior-Superior(i-s)—Below to above or lower to upper; from the trunk towards the head.

3.28 Injury—Physical disturbance, damage, or destruction to a biological structure which impairs or prevents its normal functioning.

3.29 Injury Level—A rating of a trauma's severity relative to its threat to life or degree of physical or functional impairment (cf: Abbreviated Injury Scale).

3.30 Injury Criterion—A numerical relationship between measurable engineering parameters and injury level.

3.31 In Situ—In its normal location in the body.

3.32 Intervertebral Disc—Circular pads of fibrous cartilage situated between adjacent vertebrae in the backbone.

3.33 In Vivo—Within the living body.

3.34 Laceration—A wound made by cutting or tearing.

3.35 Larynx (pl. Larynges)—The muscle/cartilage structure at the front of the neck.

3.36 Lesion—Any bodily disfunction or damage.

3.37 Ligament—A band of tissue that connects bone or supports viscera.

3.38 Loading—See Measurable Engineering Parameter.

3.39 Mandible—The bone of the lower jaw.

3.40 Maxilla—The bone which forms the central portion of the upper jaw.

3.41 Measurable Engineering Parameter—Physical behavior of a system detectable by instrumentation which describes the externally applied environment to, or the structural response or, the system or its surrogate. (Examples of measurable engineering parameters are force, acceleration, strain, and pressure).

3.42 Mobilized—Made free to move. (See Surgically Mobilized).

3.43 Occipital Condyles—Rounded prominences on each side of the base of the skull which articulate with the uppermost vertebra of the neck.

3.44 Occiput—The bone forming the rear and lower rear portion of the skull.

3.45 Patella—Knee cap.

3.46 Pneumothorax—An accumulation of air or gas in the sac surrounding the lungs.

3.47 Posterior—Rear.

3.48 Process—A prominence or projection on a bone.

3.49 Rotation—When applied to neck motion refers to the no gesture of the head.

3.50 Sagittal—A plane or section dividing the body into right and left portions.

3.51 Spinous Process—A projection of the rear on a vertebra.

3.52 Sternum—Breastbone.

3.53 Subclavian Arteries—Two of the four major blood vessels arising from the top of the heart; the subclavian arteries pass under the clavicles and supply blood to the upper body.

3.54 Subdural Hematoma—Bleeding between the two layers surrounding the brain.

3.55 Superior—Above.

3.56 Supracondylar—Situated above (superior to) a condyle.

3.57 Surgically Mobilized—Separated by surgery from its surrounding tissues but leaving connecting blood vessels intact.

3.58 Suture—A joint in which the opposed bone surfaces are closely united.

3.59 Symphysis—A line of union; a type of joint in which the opposing bones are firmly united by cartilage.

3.60 Temporal Bones—Two bones which make up the lower sides of the skull.

3.61 Temporo-Parietal—Side of the skull.

3.62 Tendon—A fibrous cord by which a muscle is attached to a bone.

3.63 Thorax—Chest.

3.64 Thyroid Cartilage—A wishbone shaped stiff tissue located in the upper portion of the neck.

3.65 Tibia—The larger of the two long bones of the lower leg.

3.66 Tolerance Level—The magnitude of loading which produces a specific injury level.

3.67 Tolerance Specification—An impact level which is taken as the maximum (or minimum) allowable condition for design purposes.

3.68 Trachea—The windpipe.

3.69 Trauma—See Injury.

3.70 Vertebra—One of the thirty-three bones of the spinal column.

3.71 Zygoma—Cheekbone.

4. Introduction To Biomechanics

4.1 Test Subjects—Of necessity, human tolerance levels must be inferred by indirect means such as testing volunteers (below their injury level), cadavers, or anesthetized animals. Each of these subjects has advantages and shortcomings which influence the applicability of the resultant data.

4.1.1 HUMANS—Volunteers provide the primary source for determining the effects of muscle tone and pre-bracing on the dynamic behavior of humans. Volunteers can also provide some information on the upper boundary of the "no injury" tolerance level. However, true tolerance levels cannot be determined with volunteers since they cannot be tested into the injury range. A further disadvantage is that volunteers are usually young, robust males whose pain and injury tolerance is apt to be considerably higher than that of the general population. Finally, the muscle bracing which volunteers sometimes employ can have an important effect at low levels which cannot necessarily be extrapolated to higher levels.

4.1.2 CADAVERS—Cadavers are normally employed when testing is undertaken at severity levels which would be injurious to volunteers. Cadavers are logical candidates as test subjects since they retain geometric similarity to living humans and many of their structures retain a strength similarity as well. This latter aspect is highly dependent on the treatment of the cadaver and the time duration since death. Recognition of these factors has led to extensive changes in cadaver testing techniques in recent years in an effort to make such test results more representative of living human response. These changes include the use of unembalmed cadavers, inflation of the lungs, and pressurization of portions of the vascular system with dye solutions to assist in trauma diagonsis. It is generally accepted that the mechanical strength of most living human body tissues decreases with age. Consequently data obtained from tests of cadavers of the elderly are likely to be conservative relative to the general population. Other potential shortcomings of cadaver testing revolve around their lack of muscle tone, and differences in some body properties from those of the living.

4.1.3 ANIMALS—Animal testing is generally employed to study the mechanisms of trauma since animals provide the only functioning physiological systems which can be subjected to severe impacts. They also provide the only known bridge for examining the relationships between living and dead subjects. Thus animals may provide the only possibility for evaluating the usefulness of cadaver testing. Unfortunately, the results of animal tests cannot, as yet, be quantitatively scaled with confidence to determine human tolerance levels due to the size, shape, and structual differences between animals and humans.

4.2 Application of Biomechanical Data

4.2.1 HUMAN SURROGATES—The behavior of the human surrogate is an important consideration when the biomechanical data of Section 5 are applied to automotive testing. To be of value, the surrogate must be sufficiently human-like so that its performance will be indicative of human behavior under similar circumstances. The problems of achieving such correlations are discussed in Section 6 of this report.

4.2.2 DETERMINATION OF TOLERANCE LEVELS—A comprehensive discussion of the factors involved in the determination of human tolerance levels is beyond the scope of this report. Indeed, such specifications are beyond the state-of-the-art in biomechanics except perhaps for a few academic situations. There are several difficulties which prevent a ready establishment of human tolerance levels. First, there are differences in judgement as to the specific degree of injury severity that should serve as the tolerance level. Second, large differences exist in the tolerances of different individuals. It is not unusual for bone fracture tests on a sample of adult cadavers to show a three-to-one load variation. Presumably, variations of at least this magnitude exist in the living population. Finally, most tolerance levels are sensitive to modest changes in the direction, shape and stiffness of the loading source. The above considerations indicate that complete and precise definitions of human tolerance levels will require large amounts of data based on controlled statistical samples. Only in this way can the influence of age, size, sex, and weight be comprehensively assessed and only in this way can mean loads and statistical measures of scatter be linked to specific tolerance levels.

In the interim, it is necessary to employ various tolerance measures in the development and evaluation of safety features. Probably the most widely used of such measures is the tolerance specification. This is an impact level taken somewhat arbitrarily as a boundary condition for design purposes. The tolerance specification should not be confused with the tolerance level which is the magnitude of loading that produces a specific degree of injury. As explained above, complete definitions of tolerance levels properly should be statistical measures relating probabilities of injury and degrees of injury to impact histories.

4.3 Biomechanical Materials—The body is composed of hard and soft materials which can occur together in composite body structures such as the rib cage and vertebral column. The presence of soft tissue as a bone connector allows for large structural deflections.

4.3.1 BONE—In addition to being non-homogeneous and anisotropic, a given bone often varies in shape from individual to individual. Therefore, it is not generally convenient to employ conventional stress analysis techniques for estimating the strength of a bone from its material properties. To overcome this difficulty, bones are generally tested in situ to determine their load carrying capacity as a structure. As with any structure, a variety of failure modes is usually possible depending on the distribution and location of the applied forces and, for impact situations, time duration effects may be important as well. Accordingly it is important to understand the mode of load application (that is, torsion, bending, shear) for the situations presented in Section 5; these tolerance levels should only be applied under similar conditions.

The bones of the skull and knee cap are uniquely sensitive to punch through (bearing load) failures. This is due to their anatomical construction as well as to their physical prominence and lack of soft tissue covering to provide a padding effect. The bones of the skull and knee cap are of a sandwich construction. Their innermost and outermost layers are shells of compact bone which embrace a middle zone of porous bone between them. Excessive bearing loads can punch through the outer shell at force levels that would not cause failure of the overall bone structure. Examples of this bearing load effect are given in Section 5.

4.3.2 SOFT TISSUES—The development of injury criteria for soft tissue trauma is an extraordinarily complex subject which is only in its early development stage. Progress in this field is likely to be slow for the following reasons:

a. A wide variety of possible injury mechanisms exist.
b. Small differences in the location or level of injury can have vastly different consequences to the injured person.
c. The capability to analyze and model the organs is very limited.

4.3.2.1 *Skin*—Skin has been studied more than any other soft tissue insofar as automotive collision trauma is concerned. The state-of-the-art in assessing skin injuries is summarized in SAE Information Report J202. One test procedure is to expose a synthetic skin material to a standardized impact test and then to evaluate the injury level either by subjective observation or measurement of the resultant damage to the synthetic material. This appears to be a practical method to evaluate skin injury levels when the multiplicity of skin injury mechanisms are considered. Skin trauma includes:

a. Avulsion (tearing away).
b. Contusion (bruising from direct impact).
c. Laceration (cutting).
d. Puncturing.
e. Splitting.
f. Abrasion.

4.3.2.2 *Internal Soft Tissues*—Internal soft tissues are vulnerable to all of the above types of trauma except abrasion. In addition, they can be injured by excessive displacement which may detach an organ from its vascular or ligamentous connections. In the brain, rapid displacement may result in injury due to cavitation. Unfortunately, little quantitative data exist on force, penetration, or displacement levels that are injurious to soft tissues. No synthetic internal organs are currently in common use for impact testing.

5. ***Data—***The human body can be subjected to a broad variety of trauma caused by a number of injury mechanisms; certain of these predominate for each zone of the body. Therefore, this information report discusses each body zone separately. They are considered in body order from head to legs.

5.1 Fracture Loads for the Cranium

5.1.1 FRACTURE MODES—The bones of the cranium are not homogeneous throughout their thickness, but are of a sandwich construction. The sandwich consists of a core of cancellous (low density) bone between two layers of compact (high density) bone. This arrangement allows two different types of bone fractures to occur, each arising from a different failure mode.

5.1.1.1 *Linear Fractures (failure of the structure as a whole due to bending stresses)*—When the impact is well distributed, the skull will be bent inwardly at the site of the blow, and outwardly in some regions remote from the blow. The tensile stresses (or strain energy densities according to one theory) arising in the latter regions can precipitate a crack. The crack usually originates at some point of stress concentration and propagates towards the site of the blow along an essentially direct path. The bone on each side of the crack remains in alignment in this type of fracture. A linear fracture is not life threatening per se since it does not in itself precipitate brain injury. However a linear skull fracture is a cause for concern since the integrity of the skull has been lost.

5.1.1.2 *Depressed Fractures (localized failure of a cranial bone due to concentrated forces)*—If the impact force is sufficiently concentrated, it may break through the structure locally even though the magnitude of the force might be insufficient to over-stress the bone structure as a whole. A contact area of approximately 2 in^2 (13 cm^2) is considered here to represent the transition between distributed and concentrated loading. As the contact area diminishes below this threshold size, depressed fractures are likely to occur as a result of localized stresses at the impact site. This produces a cave-in mode of failure. If the contact area is further diminished, to less than approximately 3/4 in^2 (5 cm^2) the depressed fracture takes the form of a clean punch-through with a hole size which matches the size of the struck object. This behavior is thought to be due to two concurrent failures: compression of the core of cancellous bone and shearing of the compact bone. Both the cave-in and punch-through types of depressed fractures result in an inward displacement of the bone which can lead to mechanical impingement against the brain and allow the entry of foreign bodies. Depressed fractures are potentially life threatening injuries.

5.1.2 FRACTURE DATA—Table 1 presents a summary of fracture load data for the cranium. Some of the anomalies found there can be explained by considering the impactor shape and the cranial bone properties; however, most are likely due to the large variations that are inherent in any cadaver population. The likelihood of inconsistencies is compounded by the small number of test specimens employed in many of the test series cited. These considerations suggest the need for caution in the use of this table.

TABLE 1—DYNAMIC FRACTURE FORCES FOR CRANIAL BONES RIGID SURFACE IMPACTS

Type of Impact Surface	Fracture Forces(1) Mean lb	Fracture Forces(1) Mean N	Fracture Forces(1) Range lb	Fracture Forces(1) Range N	Sample Size	Fresh or Embalmed	Ref
Frontal Bone							
flat plate(2)	1430	6360	880–2650	3910–11 790	12	fr	1
flat plate	1440	6400	1220–1770	5420–7870	6	em	2
longitudinal surface of cylinder							
1 in (2.5 cm) rad. aligned transversely	1260	5600	950–1650	4220–7340	7	em	3
1 in (2.5 cm) rad. aligned sagittally	1600	7120	940–2000	4180–8900	5	em	2
5/16 in (0.79 cm) rad. aligned transversely	1230	5470	700–1730	3110–7700	5	em	3
sphere, 8 in (20.3 cm) rad.	1180	5250	830–1530	3690–6810	5	em	2
small area flat surfaces							
1 1/8 in (2.9 cm) dia	1130	5030	848–1600	3770–7120	5	em	4
1 1/8 in (2.9 cm) dia	1390	6180	980–1990	4360–8850	5	fr	4
1 1/8 in (2.9 cm) dia	1310	5830	930–2220	4140–9880	7	em	5
0.61 in (1.55 cm) dia	1710	7610	920–2200	4090–9790	5	em	6
0.43 in (1.09 cm) dia	1030	4580	470–2000	2090–8900	5	em	6
small area rounded surface							
0.67 in (1.70 cm) dia(2)	1000	4450	620–1820	2760–8100	6	fr	1
Temporo-Parietal Bones							
flat plate (2)	1140	5070	770–1760	3430–7830	13	fr	1
flat plate	1910	8500	1050–3360	4670–14 950	7	em	2
small area flat surfaces							
1 1/8 in (2.9 cm) dia	846	3760	550–1330	2450–5920	7	fr	5
1 1/8 in (2.9 cm) dia	702	3120	302–1330	1340–5920	8	em	5
0.61 in (1.55 cm) dia	1290	5740	500–2200	2220–9790	10	em	6
0.43 in (1.09 cm) dia	780	3470	140–1500	620–6670	10	em	6
small area rounded surface							
0.67 in (1.70 cm) dia(2)	766	3410	400–1100	1780–4890	7	fr	1
Occiput Bone							
small area rounded surface							
0.67 in (1.70 cm) dia(2)	1440	6410	1150–2150	5120–9560	5	fr	1
Padded Surface Impacts							
Frontal Bone							
rubber padded rigid surface							
flat sheet, 90 durometer	2530	11 260	1200–3400	5340–15 100	7	em	2
1 in (2.5 cm) rad. cyl. 90 durometer aligned sagittally	1650	7340	1100–1960	4890–8720	6	em	2

1. These forces produced a variety of fracture patterns: see the source material referenced for a description of the fractures produced by each surface.
2. Static tests.

5.2 Brain Injury—The brain can be injured by processes other than the fracture/impingement mechanism described above. Excessive acceleration, by itself, can cause brain injury through a variety of effects, none of which are completely understood. Relative motion between the brain and the skull can induce a wide range of debilitating effects; the periphery of the brain can be contused, the blood vessels leading from the brain to the skull can be ruptured, internal brain matter can be sheared by relative motion between its parts, and the brain stem can be distorted by extrusion through the opening at the base of the skull. Finally, excessive tensile stresses can occur independent of any large brain displacement. This usually takes place opposite the impact site and can disrupt a variety of brain functions depending on its location. Little is known about the effects of multiple or long duration impacts.

5.2.1 CONCUSSION—In 1966, the Committee of the Congress of Neurological Surgeons defined brain concussion as:

"A clinical syndrome characterized by immediate transient impairment of neural function such as alteration of consciousness, disturbances of vision, equilibrium, etc., due to mechanical forces."

Concussion is usually a fully reversible injury. It has been widely studied for a number of reasons:

a. It is by far the most prevalent brain trauma.
b. Concussion is usually the first functional impairment of the brain to occur as the severity of head impact increases.
c. It accompanies 80% of all linear skull fractures (however the vast majority of concussions occur without skull fracture).
d. It is reproducible in experiments with animals whereas other brain injuries are not.

For these reasons, early brain injury studies were based on analysis of concussion rather than the more complex injury mechanisms described previously.

5.2.2 HISTORICAL DEVELOPMENT—Three different aspects of the gross skull motion have been suggested as being correlated to concussion:

a. Rotational acceleration.
b. Translational acceleration.
c. Flexion-extension of the upper cervical cord during motion of the head-neck junction.

Only the first two phenomena have received quantitative appraisals and are discussed here.

5.2.2.1 Rotational Acceleration—Leaders in this field have been Holbourn (Ref. 7), Gurdjian, et al (Ref. 8), Ommaya, et al (Ref. 9), Unterharnscheidt, et al (Ref. 10), Gennarelli, et al (Ref. 11), and Hirsch, et al (Ref. 11). In more recent years, most of the research into the effects of rotation have been conducted on animal brains, in vivo or in isolation. Hirsch's group has attempted to establish injury criteria and tolerance levels for rotational acceleration. Based upon results of experiments with several types of monkeys, they have employed scaling laws to apply tolerance levels to the human.

5.2.2.2 Translational Acceleration—The first version of the Wayne State University Concussion Tolerance Curve (Figure 2) was proposed by Lissner, et al in 1960 (Ref. 13). The abscissa is the duration of the effective part of the pulse spanning the principal impact. The ordinate is effective acceleration which is the average $a\text{-}p^2$ acceleration of the skull measured at the occipital bone for the principal part of the impact of the forehead against plane, unyielding surfaces. The curve was derived from the following observations:

a. It was observed clinically that linear skull fracture is usually associated with unconsciousness or a mild concussion (Gurdjian, et al. (Ref. 14)).

2. Anterior-posterior or front to back.

b. The acceleration levels and pulse durations necessary to cause skull fractures in cadaver heads were measured in free fall impacts against a rigid surface. These results were considered to approximate the human tolerance level for concussion from the correlation noted in item (a) above. The fracture data provided points for the curve in the range of 0.001–0.006 s.
c. Experimental animals were concussed by air pressure pulses of varying magnitudes and durations applied directly to the membranous covering of the brain (Gurdjian, et al. (Ref. 15)).
d. The pressure pulses measured in the parietal and temporal regions of cadaver heads in drop tests (Lissner, et al (Ref. 13)) and Gurdjian, et al. (Ref. 16)) were compared with the animal data in item (c), and the corresponding cadaver acceleration measurements were used to provide data points for the concussion curve between 0.006 and 0.010 s.
e. The long-duration end of the curve, with the asymptotic value of 42 G, was obtained from whole body volunteer data reported by Stapp (Refs. 17, 18). Patrick, et al, considered this value to be too low, since other volunteers had survived frontal crash simulations exceeding 45 G. They recommended that the value of the asymptote be raised to 80 G for padded impacts that avoid concentrated loads (Ref. 18). The resulting curve (Figure 2) became the accepted version of the Wayne State Tolerance Curve and is the basis of most current head injury criteria including the original U.S. Federal Motor Vehicle Safety Standard head impact specification (FMVSS 201) (Ref. 20).

Figure 1

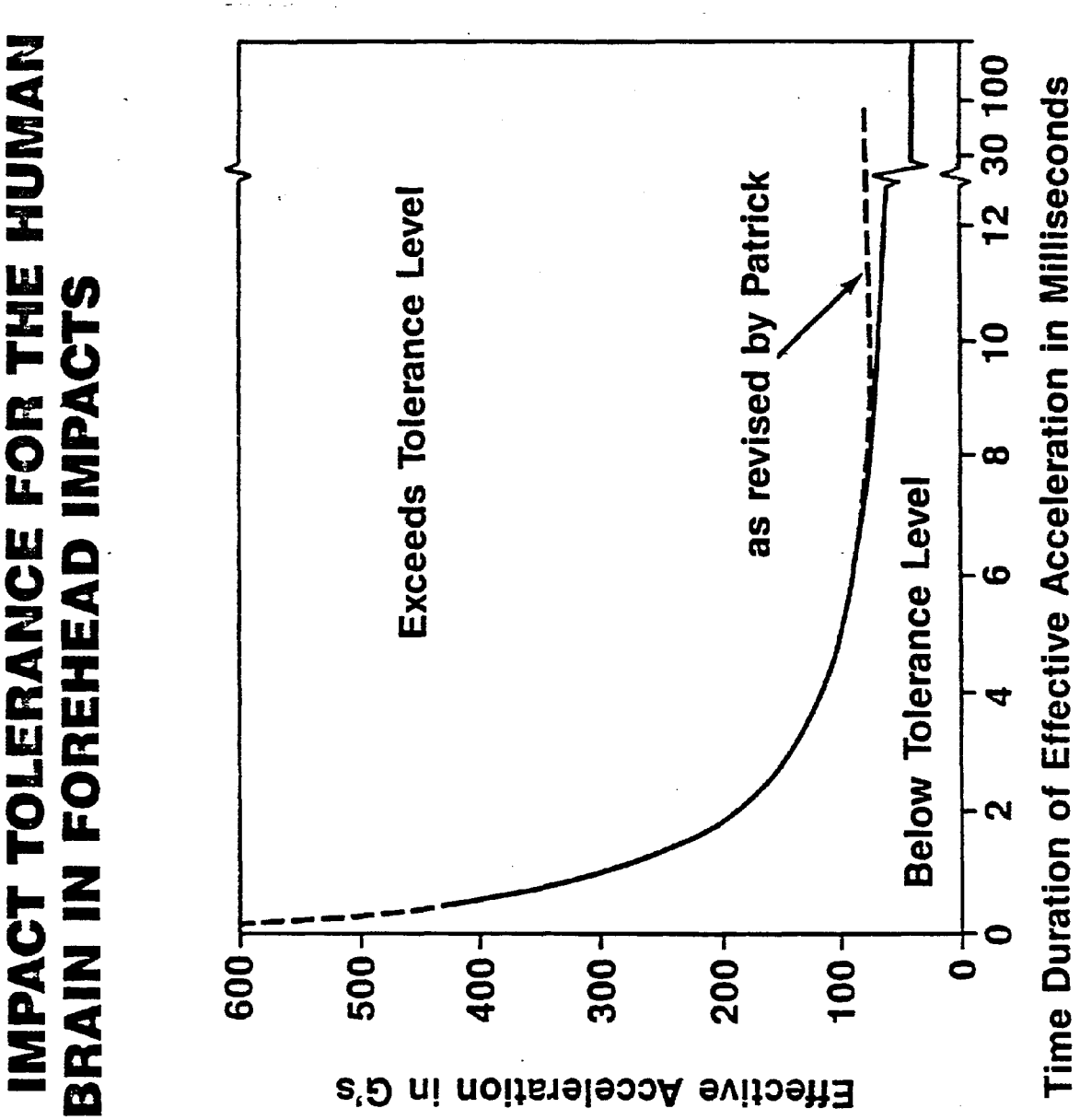

FIGURE 1—

Ono, et al, (Ref. 21) concluded that their results supported the Wayne State Concussion Tolerance Curve given in Figure 1. This conclusion was based on extensive microscopic examination of brain tissues and physiological measurements following translational and rotational impacts to the heads of sixty-three monkeys, and drops of fifteen human cadavers' skulls.

5.2.3 INJURY CRITERIA AND TOLERANCE LEVELS—The two principal criteria of brain injury are the Severity Index (SI) and the Head Injury Criterion (HIC). Numerous additional indices of brain injury have been proposed. Most of these are summarized in Ref. 22.

5.2.3.1 *Severity Index*—The WSU Tolerance Curve is difficult to apply to complex acceleration-time pulses because of uncertainties in determining the effective acceleration and time. To overcome this problem, Gadd (Refs. 23, 24) devised a weighted impulse criterion for establishing a Severity Index (SI):

$$SI = \int_O^T a^n dt \qquad \text{(Eq. 1)}$$

where:

a = acceleration in G's
n = weighting factor, 2.5 for head impacts
T = pulse duration
t = time in seconds

The weighting factor of 2.5 is primarily based on the slope of the straight-line approximation of the Wayne State Tolerance Curve plotted on log-log paper between 2.5 and 50 ms. A review of the mathematical derivation of the Severity Index by Versace (Ref. 27) details the relationship between the Wayne State Curve and the Severity Index. Gadd proposed a tolerance value of 1000 as the threshold of concussion for frontal impact (Ref. 23). This tolerance value was mandated in early versions of FMVSS 208; however, it specified that the Severity Index was to be calculated using the resultant acceleration measured at the CG of the head instead of the uniaxial acceleration measured on the occiput in the direction of the blow as was used by Gadd.

For distributed or non-contact blows to the head, Gadd (Ref. 25) has indicated that an SI value of 1500 would be an appropriate concussion tolerance level. Gadd cited the fact that Stapp experienced an acceleration pulse which equated to a true biaxial head exposure estimated to have reached a Severity Index of 1500. This occurred in a rocket sled run in which 45 G was measured on the seat. There was no brain injury in this exposure although retinal hemorrhages occurred.

Some success has been reported in employing the Severity Index concept to reduce brain injuries occurring to football players. Seventy-three to one hundred percent of the brain injury fatalities reported annually in this sport have been subdural hematomas. Beginning in 1970–1971, football helmets were designed to attenuate head impacts to an SI of less than 1500 in a simulation of a severe football head impact. The influence of this impact criterion is shown by a 50% reduction in fatality incidents (normalized) when comparing the post 1971 seasons to the preceding equivalent period (Ref. 26).

5.2.3.2 *Head Injury Criterion (HIC)*—Versace (Ref. 27) examined the relationship between the Wayne State Curve and the Severity Index. In response to this, a new parameter, the Head Injury Criterion (HIC) was defined by NHTSA as:

$$HIC = \left\{ (t_2 - t_1) \left[\frac{1}{t_2 - t_1} \int_{t_1}^{t_2} a(t) dt \right]^{2.5} \right\} \max \qquad \text{(Eq. 2)}$$

where t_1 and t_2 are the initial and final times (expressed in seconds) of the interval during which HIC attains a maximum value and a(t) is the resultant acceleration (expressed in G) measured at the head CG. The HIC replaced the SI in later versions of FMVSS 208 with a HIC value of 1000 being specified as the concussion tolerance level.

A point worth noting is a study by Hodgson and Thomas (Ref. 28) which concluded that the HIC interval (t_2 - t_1) must be less than 15 ms in duration in order to pose a concussion hazard even if the HIC value exceeds 1000. This finding resulted from an examination of events for which the concussive outcomes were known or could be inferred.

5.2.3.3 *Mathematical Models of the Brain*—Some researchers in brain injury have sought insights into its behavior by developing mathematical models of the brain and/or skull. Early investigators in this field employed continuum models. Much of this work is reviewed in Ref. 29. These simple models proved to be unsatisfactory and attention turned to finite element formulations. Ward and Thompson developed one of the more advanced models of this type (Ref. 30). More recently Ward et al proposed a brain injury criterion based on the intracranial pressure calculated from the model's response to input accelerations (Ref. 31).

5.2.3.4 *Lateral Tolerance of the Brain*—All of the preceding discussion is based on data obtained from head impacts in the a-p direction. Some lateral studies employing cadavers and primates have been reported by Stalnaker, et al (Ref. 32). They concluded that the threshold of irreversible closed skull brain injury to humans occurred when the translational head acceleration reached a peak of 76 G with a pulse duration of 20 ms.

Got, et al performed twenty-two lateral drop tests employing cadavers with pressurized brain arterial systems (Ref. 33). Seventeen of these specimens were helmeted, three were unhelmeted and struck padded surfaces, and two were unhelmeted and struck a rigid surface. Sixteen of these tests were considered to have produced useful results, with HIC values ranging from 900 to over 2000. For HIC values equal to or below 1500 (10 cases) two specimens exceeded an AIS injury level of 3; for HIC values equal to or below 1000 (two cases) one specimen received an AIS injury level of 0 and the other an AIS injury level of 2. It should be noted that the brain damage found with the use of brain pressurization procedures employing liquid dyes are typically arterial ruptures which are more serious injuries than the reversible concussion on which Figure 1 is based. The venous system was not pressurized reducing the chance of detecting a failure in its vessels. It should also be noted that neurophysiological damage cannot be detected in cadaver experiments.

Melvin et al (Ref. 34) investigated lateral impacts to unembalmed cadavers against rigid and deformable structures. They found, for head impacts against rigid walls, that brain damage of AIS 4 or greater began to occur at head impact speeds of 20 mph (33 km/h).

Nahum et al performed lateral impact tests to the heads of five cadavers using a padded impactor (Ref. 35). Arterial pressurization was employed with post test dissection showing subarachnoid hemorrhages on the lateral brain surfaces. HICs ranged from 1340 to 5246 with a mean of 2930.

5.3 Strength of Facial Bones—The principal facial bones are the mandible (lower jaw), maxilla (upper jaw), and the two zygomas (cheekbones). All are prominent and can be struck in a variety of locations and from a variety of directions. In addition, these bones can be loaded individually or collectively depending on the size, shape, and conformability of the impacted surface. The literature on facial bone fracture is limited, but the far greater part of it deals with the strength of the individual bones. The typical impactor employed was flat, circular, of 1 in^2 area (6 1/2 cm^2) and covered with little or no padding. Test impacts were usually delivered to the most prominent feature of the bone and essentially normal to it. A summary of facial bone fracture data is available in Table 2.

5.3.1 ZYGOMA—Four studies are available in which a 1 1/8 in (2.9 cm) diameter impactor was used to strike the zygoma. Three of these studies employed blows to the frontal portion of the zygoma (near its junction with the maxilla) while the fourth study addressed the mid-arch. The results of these four investigations were similar and their findings can be summarized by the results reported by Nahum, et al. (Ref. 36).

a. The minimal tolerance load was 200 lb (0.89 kN); their recommended level for a clinically significant fracture was 225 lb (1kN).
b. Embalming did not appear to affect results for the areas studied.
c. Thickness of the overlying soft tissue played an important role.

TABLE 2—DYNAMIC FRACTURE FORCES FOR FACIAL BONES

Bone	Impactor	Fracture Forces Mean lb	Fracture Forces Mean N	Fracture Forces Range lb	Fracture Forces Range N	Sample Size	Fresh or Embalmed	Ref
zygoma [1]	[2]	386	1717	138–780	614–3470	19	both	36
zygoma[1]	[3]	374	1665	208–640	925–2850	10	both	5
zygoma	[4]	283	1259	190–374	845–1665	5	embalmed	37
zygoma	[5]	516	2297	360–756	1600–3360	7	embalmed	37
zygomatic arch	[3]	345	1535	208–475	925–2110	17	both	5
maxilla	[3]	258	1148	140–445	623–1980	13	both	5
maxilla (lower)	[2]	[6]		175–210[7]	778–934	(6)	(6)	36
mandible (symphysis)	[2]	[6]		350–400[7]	1558–1780	(6)	(6)	36
mandible (midbody)	[2]	(6)		290–325[7]	1290–1445	(6)	(6)	36
mandible (center)	[3]	697	3100	425–925	1890–4110	9	both	5
mandible (lateral)	[8]	431	1918	184–765	818–3405	9	both	5

1. Impacted near the maxillary suture.
2. Flat rigid impactor, 1 1/8 in (2.9 cm) dia covered with MetNet pad. 0.2 in (0.5 cm) thick.
3. Same as footnote b except pad thickness was 0.10 in (0.25 cm).
4. Padded impactor, 1 1/8 in (2.9 cm) dia.
5. Patted impactor, 2 9/16 in (6.5 cm) dia.
6. Not reported.
7. Lower range of fracture values.
8. Flat rigid impactor 1 x 4 in (2 1/2 x 10 cm) covered with nickel foam pad 0.2 in (0.5 cm) thick.

In another zygoma study, Hodgson (Ref. 37) explored the effect of increasing the area of the impactor. He conducted paired tests of five cadavers; the zygoma on one side of the face was struck with a 1 1/8 in (2.9 cm) diameter impactor while the opposite zygoma was struck with a 2 9/16 in (6.5 cm) diameter impactor. The average fracture loads were 283 lb (1.26 kN) and 573 lb (2.55 kN) respectively.

5.3.2 MAXILLA—The maxilla is the weakest of the facial bones when the impact is directed to the thin bone covering the maxillary sinus. Schneider, et al (Ref. 5) reported that every one of the fractures in their maxilla study was "depressed and comminuted" due to breakage of this bone shell. They conducted thirteen impacts (producing eleven fractures) with a 1 1/8 in (2.9 cm) diameter flat impactor. Their average fracture force was 257 lb (1.15 kN) and their fracture range was 140–445 lb (0.62–1.98 kN). A previous study of the maxilla by Nahum, et al (Ref. 36) had reported a range of 175–210 lb (0.78–0.93 kN) as a "clinical fracture tolerance".

5.3.3 MANDIBLE—The size and shape of the mandible presents a wide range of impact possibilities. Schneider, et al (Ref. 5) noted an indeterminacy in delivering impacts to the center of the mandible. If the blow was directed towards the cranium, and the teeth were in contact, high forces could be sustained before failure occurred at the mandibular body or its symphysis. However, if the blow was directed towards the neck, the loading was carried primarily by the condylar processes (where the jaw articulates with the skull) which failed at lower loads. In this Schneider series, fractures occurred at all three locations; the fracture force levels for the nine specimens tested range from 425–925 lb (1.89–4.11 kN) with an average value of 639 lb (2.84 kN) for the six failures obtained. A previous study (Ref. 36) had found a "clinical fracture range" of 350–400 lb (1.56–1.78 kN) for impacts to the symphysis of the mandible.

Lateral impacts to the body of the mandible have been undertaken both with a 1 1/8 in (2.9 cm) diameter impactor and a 1 x 4 in (2 1/2 x 10 cm) rectangular impactor aligned along the body. The former study obtained "lower fracture values" of 290–325 lb (1.29–1.44 kN) (Ref. 36) while the latter study produced a fracture range of 184–765 lb (0.82–3.41 kN) and an average fracture load of 431 lb (1.92 kN) (Ref. 5).

5.3.4 FULL FACE—One study is available which indicates that the facial skeleton is remarkably strong when face contact occurs against a padded, deformable surface. Daniel and Patrick (Ref. 38) conducted 22 sled tests with lap-belted cadavers in an automobile body buck; head impact speeds ranged from 9–40 mph (4–18 m/s). Their test geometry was such that the cadaver heads typically struck the top of their padded instrument panel chin first; the head then rotated forward until full-face contact occurred. No facial bone injuries were found in this series. Head a-p accelerations were all below 60 G except for the single run at 40 mph (18 m/s); here the acceleration was 165 G.

5.4 Direct Impact to the Neck—The anterior portion (front) of the neck contains two stiff tissues which are delicate and vital. These stiff tissues, the thyroid and cricoid cartilages, are found at the upper end of the airway passage in the neck; hence their collapse can obstruct airflow. The thyroid cartilage is shaped like a wishbone with a relatively blunt apex. The apex (Adam's apple) faces anteriorly. The cricoid cartilage is immediately beneath the thyroid; it is ring shaped and completely encircles the trachea.

5.4.1 MELVIN DATA—The fragility of the thyroid and cricoid cartilages is illustrated by the data in Table 3. Melvin, et al., (Ref. 39), employed a Plastechon high-speed testing machine to conduct dynamic compression tests on eight excised, unembalmed human larynges. They found that incipient cracking occurred at a mean load of 40.6 lb (181 N) for the thyroid cartilage and 55.5 lb (247 N) for the cricoid when each was loaded separately. As a part of this program, both cartilages were also loaded simultaneously to very large deflections (one-half their original dimension) through the use of a 1 1/2 in (38 mm) diameter flat plate. For this situation, the force increased to a mean level of 110 lb (490 N). It should be noted that this 50 percent deflection represents a very serious fracture level at which total collapse of the larnyx was imminent.

5.4.2 GADD DATA—Another larynx study by Gadd, et al. (Ref. 40), tested unembalmed human subjects with the larynx in situ and obained somewhat higher loads than Melvin. The Gadd program employed an instrumented drop weight of 1 in^2 (6 1/2 cm^2) area and produced marginal fractures of either the thyroid or cricoid cartilage at dynamic loads of 90–100 lb (400–450 N).

5.5 Neck Injury Due to Head Inertia Loading—In automobile collisions, neck injuries can occur as a result of its bending from head inertial loading. When the torso is violently accelerated (or decelerated), large, potentially injurious neck loads and deflections are generated by the inertia of the head.

Neck bending can occur in any direction. In medical terminology, backward bending of the neck is called extension; forward bending of the neck is termed flexion, sidewards bending of the neck is called lateral flexion; the "no" gesture of the head is termed rotation.

TABLE 3—DYNAMIC FRACTURE LOADS FOR THE THYROID AND CRICOID CARTILAGES

Cartilage	Dynamic Fracture Loads Mean lb	Dynamic Fracture Loads Mean N	Dynamic Fracture Loads Range lb	Dynamic Fracture Loads Range N	Nature of Fracture	Ref
excised thyroid	40.6	180	14–85	62–377	incipient cracking	39
excised cricoid	55.5	248	35–68	156–302	incipient cracking	39
thyroid and cricoid loaded simultaneously	100	490	76–182	337–810	imminent total collapse	39
thyroid in situ	90–100	440–445			marginal fracture	40
cricoid in situ	90–100	400–445			marginal fracture	40

5.5.1 NECK STRUCTURE—The neck skeleton consists of seven cervical vertebrae. These vertebrae are generally referred to by number in order from top to bottom as C-1, C-2, etc. No two cervical vertebrae are identical; however, C-3 through C-7 are quite similar to one another. Adjacent vertebral bodies are separated by cartilagenous tissues called intervertebral discs. Vertebral articulations are stabilized by fibrous connecting tissues called ligaments. These ligaments also limit the degree of relative motion between the vertebrae.

Relative movement of the cervical vertebral column and the head is accomplished through muscle pairs which are attached to the skull, the individual vertebrae, and the torso through tendons. These pairs, which are symmetric on the right and left sides of the body, respond in various group actions to produce the desired movement of the head and neck. Muscle pairs which produce voluntary flexion are the ones which resist extension, and vice versa.

The muscles lying behind the head/neck are more massive than those lying in front; in addition, the former are located further from the head-neck pivot (the occipital condyles). Consequently, larger moments can be developed for resisting flexion than for extension. Also, a lower resultant muscle force level is required to produce the same magnitude of resisting bending moment in flexion than would be required in extension.

5.5.2 INJURY MECHANISMS

5.5.2.1 *Hyperextension Injuries and Associated Mechanism*—The rear-end collision accounts for most of the diagnosed neck injuries that occur to vehicle occupants. The resultant neck lesions are generally classified as hyperextension trauma and include symptoms such as localized neck pain, pain radiating to the shoulders, vague aches, discomfort, and vertigo due to strained muscles, damaged ligaments, injured articular joints, or fractures of various parts of the cervical vertebrae. The involvement of the cervical vertebrae, joints, connecting ligaments, and muscles in a rear-end collision environment can be qualitatively analyzed but are difficult to quantify.

If the head is turned to one side at the onset of a rear-end collision, the neck ligaments will be prestrained and less articulation of the neck will be required to produce high resistive forces. Consequently, there will be less time available for the neck muscles to respond to aid in accelerating the head, placing a greater burden on the ligaments. For this condition, the neck can be more susceptible to injuries.

5.5.2.2 *Hyperflexion Injuries and Associated Mechanisms*—Hyperflexion neck injuries to lap/shoulder belted occupants have not been reported with any degree of frequency in field accident studies. A study by Vazey and Holt (Ref. 41) of fatalities to car occupants wearing lap/shoulder belts indicated that only two out of 136 fatalities were due to neck injuries. In these two cases, the occupant compartments were severely compromised. The involvement of the shoulder harness in producing these two neck injuries is doubtful. In contrast, Schmidt et al (Ref. 42) conducted a series of one hundred simulated frontal collision sled tests using lap/shoulder belted cadavers as vehicle occupants. Sled impact speeds of 30, 40 and 50 km/h were used. The deceleration-time curve was trapezoidal with plateau deceleration levels ranging between 16.9 to 25.6 G's. The cadavers were unembalmed with an age distribution at time of death ranging from 12 to 83 years. While the most frequent cadaver damage observation was rib fracture, 46 of the 100 cadavers had neck damage with most of this damage being concentrated at the level of C-7 and T-1.

There are a number of possible explanations for the difference in frequency of field and experimental neck flexion damage. The neck structure of the unembalmed cadaver is flaccid. Its neck muscle cannot transmit any significant load even when the neck is hyperflexed. All the neck loads must be transmitted by the bony vertebrae, the intervertebral discs and the surrounding joint ligaments. In the living human, neck muscles can transmit loads, sharing the load distribution with other neck structures. The implication of this difference in the load carrying structures of the cadavers and the human neck is that while the cadaver will mimic the neck damage patterns of the human, the cadaver damage will occur at lower collision severity levels. On the other hand, published field accident data of neck injury may not represent the actual frequency of neck injuries since detailed autopsies of the neck are not routinely performed. Also, in the more severe frontal collision environments, the head of the belted occupant may impact a part of the forward interior. This is particularly true of the driver. In such cases, the head load causes a redistribution of the neck loading which could reduce the potential for neck injury.

Shear forces in the neck are important in flexion prior to the chin contacting the chest. For the vertebrae C-3 through C-7, there are bone-to-bone interlocking joint surfaces and ligaments to carry these shear forces as the neck is flexed. This is not the case for the upper neck joints (occipital condyles/C-1 and C-1/C-2). Here the ligaments must carry the shear loads. These upper joints are, therefore, the most likely to be injured by shear.

When the chin contacts the chest, a redistribution of the loading occurs. Chin-chest contact causes a lower force level to be developed in the posterior muscles for the same magnitude of resisting bending moment. In addition, the force on the chin has a component which is parallel to the shear force developed by the neck and aids in decelerating the head. Transfer of loading from the ligaments of the neck to the chin reduces the shear load transmitted between the head, C-1, and C-2 and reduces the probability of injury in these areas.

As the neck flexes, the front portions of the intervertebral discs are compressed. Lesions to the discs may result if the compressive forces become sufficient. Also, the anterior portions of the vertebral bodies may be fractured. The ligaments posterior to the articular surfaces can be torn during hyperflexion. In particular, the ligaments joining adjacent spinous processes are prime candidates for lesions since they undergo the greatest elongation. The ligamentous and muscle loads may fracture the spinous processes or parts of the vertebrae surrounding the spinal cord.

5.5.2.3 *Lateral Flexion Injuries and Associated Mechanisms*—Lateral flexion injuries occur less frequently than the other two types of neck injuries. Usually in a lateral (side impact) collision, severe lateral flexion of the neck does not occur. For a far side collision, the upper torso is accelerated but may be free to rotate towards the impacted side, minimizing the neck forces required to accelerate the head. For a near side collision, the torso is accelerated upright, but the head usually impacts the side door window or upper side structures minimizing the neck forces. If severe lateral flexion should occur, ligamentous injury and/or fractures of the articular processes of the vertebrae may be found at the C-5 to C-7 level.

5.5.3 NECK STRENGTH—To obtain measures of each injury mechanism which was discussed previously would be a difficult, if not an impossible task. First, volunteers cannot be exposed to injury-producing environments; second, relevant in vivo measurements cannot usually be made. Consequently, indirect approaches must be used to obtain data which can be related to the overall strength of the neck.

Three approaches have been used to obtain neck strength data. Static strength tests on necks have been conducted with volunteers resisting static loads applied to their heads. Dynamic tests have been conducted where volunteers have been subject to controlled, non-injurious acceleration environments. In these latter tests, the torso is restrained and the head is accelerated by the neck. A third approach utilizes human cadavers in a similar manner to the dynamic volunteer tests, except that the severity of exposure can be increased until physical damage to the neck structure is produced.

In dynamic neck tests, it is common practice to measure accelerations of the head and the angular position of the head relative to the torso; in static tests the usual measurement is the force applied to the head. Investigators have noted deficiencies in relating injury severity to these measurements. For the static tests, the applied head load does describe the force level the neck must resist, but does not directly define the resisting bending moment the neck must develop. The same is true of the accelerations measured in the dynamic tests. In an effort to minimize these deficiencies, Mertz and Patrick (Ref. 43) developed a method for calculating the resultant reactions developed between the top of the neck and the base of the skull (occipital condyles) for both the static and dynamic approaches. This method allows direct comparisons to be made of static and dynamic neck reactions.

The angular position of the head relative to the torso can be used as a measure of the severity of neck bending. However, it should be noted that the neck can be injured without exceeding its static angular range of motion. In addition, when the neck is flexed to an extreme of articulation, relative angular position becomes a poor measure of potential injury because a small increase in articulation, which is difficult to measure accurately, will produce large increases in neck loads. Measures of the neck loads may be a better indicator of injury potential.

5.5.3.1 *Static Strength of the Neck*—Mertz and Patrick (Refs. 43, 44) and Patrick and Chou (Ref. 45) have conducted tests on volunteers to determine the neck's reaction on the head for statically applied loads to the head. The principal results of these studies are summarized in Tables 4 and 5. Table 4 is a summary of the maximum static bending moments developed at the occipital condyles for various loading conditions. The maximum shear and axial forces that were observed are given in Table 5.

TABLE 4—MAXIMUM STATIC BENDING MOMENT DEVELOPED AT THE OCCIPITAL CONDYLES BY VARIOUS VOLUNTEERS FOR VARIOUS LOADING CONFIGURATIONS (AIS = 0)
BENDING MOMENT DEVELOPED AT OCCIPITAL CONDYLES
ft-lb (N·m)

Neck Position	Resist Flexion-0 deg	Resist Extension-180 deg	Resist Lateral Flexion-90 deg	Resist Flexion-45 deg	Resist Flexion-135 deg
Normal	37.0 (50.2)	15.0 (20.3)	35 (47.5)	40.5 (54.9)	24.0 (32.5)
Flexed[1]	30.0 (40.7)	28.0 (38.0)	45.5 (61.7)	46.0 (62.4)	40.0 (54.2)
Extended[2]	29.5 (40.0)	17.5[3] (23.7)	38.0 (51.5)	34.5 (46.8)	27.0 (36.6)

NOTE—These values are not necessarily upper bounds of tolerable neck bending moments. Tests could have been terminated for reasons other than reaching a limit on forces producing the resistive bending moment. For example, the moment arm could be progressively decreased as the neck bends due to increasing load. Thus, the magnitude of the applied load could increase, but the resistive bending moment could decrease.

1. Initial head position toward the applied load.
2. Initial head position away from the applied load.
3. Value taken from Ref 43.; all other values taken from Ref. 45.

TABLE 5—MAXIMUM STATIC FORCE REACTIONS DEVELOPED AT THE OCCIPITAL CONDYLES BY VARIOUS VOLUNTEERS FOR VARIOUS LOADING CONFIGURATIONS (AIS = 0)

Shear Force lb (N)	Shear Force lb (N)	Shear Force lb (N)	Shear Force lb (N)	Shear Force lb (N)	Axial Force lb (N)	Axial Force lb (N)
A-P	P-A	R-L L-R			Tension	Compression
0 deg	180 deg	90 deg	45 deg	135 deg		
190 (845) [1]	190 (845)[1]	90 (400)[2]	98 (436)[2]	96 (427)[2]	255 (1134)[1]	250 (1112)[1]

NOTE—These values are not necessarily upper bounds of tolerance load reactions between the head and the neck at the occipital condyles. Tests could have been terminated due to discomfort with the strapping used to apply the load to the head.

1. Values taken from Ref. 43
2. Values taken from Ref. 45

It should be noted that none of these are necessarily upper bounds of non-injury load reactions between the head and neck at the occipital condyles. Tests were usually terminated due to discomfort with the straps used to apply the load to the head. No injuries or neck pain occurred as a result of any of these loads. They are considered non-injurious neck reactions and correspond to an Abbreviated Injury Scale (AIS) rating of zero.

Gadd, et al. (Ref. 40), subjected human cadavers to static rearward and lateral neck bending loads. They noted that minor ligament injury occurred for 80 degrees of rearward neck bending and 60 degrees of lateral neck bending.

5.5.3.2 *Dynamic Strength of the Neck*—Mertz and Patrick (Ref. 43, 44) and Patrick and Chou (Ref. 45) have also conducted tests on volunteers and human cadavers to determine the neck's reaction on the head under dynamic conditions. The principal results of these studies are given in Table 6 for volunteers and in Table 7 for human cadavers. The bending moment for forward flexion includes the moment of the chin force taken with respect to the occipital condyles.

Mertz and Patrick (Refs. 43, 44) found that the resultant bending moment was an excellent indicator of neck strength. Based on their cadaver data, they suggested tolerance levels for the 50th percentile adult male. For flexion, a resultant bending moment of 140 ft-lb (190 N-m) was proposed as a lower bound for an injury tolerance level. This bending moment did not produce any discernible ligamentous damage to a human cadaver. For extension, an injury tolerance level of 42 ft-lb (57 N-m) was suggested. This level was associated with ligamentous damage to a human cadaver. However, it should be noted that the human cadaver was relatively old and, also, there may have been degeneration of the strength of the ligamentous tissue compared to living tissue. Based on these suggested bending moment tolerance levels, the neck appears to be at least three times stronger in resisting flexion than extension.

Ewing and Thomas (Ref. 46) have also conducted dynamic forward neck bending tests with instrumented volunteers. Their testing has been directed at obtaining neck response data, not tolerance data. However, for some of their more severe test conditions, they have calculated the maximum forward neck bending moments relative to the occipital condyles. Three of the volunteers developed maximum bending moments of 22.5 ft-lb (35 n·m), 33.2 ft-lb (45 n·m) and 36.9 ft-lb (50 n·m) without any pain. These values are consistent with the forward bending results of Mertz and Patrick (Ref. 43) given in Table 6 where neck pain, an AIS = 1 injury, was observed at 65 ft-lb (88.2 n·m).

Nyquist, et al. (Ref. 47), subjected an instrumented dummy (Hybrid III, Ref. 48) to simulated accident environments of lap/shoulder belted occupants. The dummy was instrumented to measure the resultant neck loadings at the interface between the head and neck. For each simulated accident condition, the type and severity of the expected neck injury were inferred from the field accident injury data. Nyquist measured a neck forward bending moment of 110 ft-lb (152 N·m) along with an a-p neck shear load of 670 lb (2.97 kN) and a neck axial tension load of 740 lb (3.29 kN) in a test condition associated with an AIS = 1 neck injury level. Environments which produced serious neck injury were not simulated in this study. The limitations of restaging field accidents are discussed in Section 6.2.2.

TABLE 6—TOLERABLE NECK REACTIONS CALCULATED AT THE OCCIPITAL CONDYLES FOR DYNAMIC VOLUNTEER TESTS

Loading Configuration	Ref	Neck Bending Moment ft-lb	Neck Bending Moment (N·m)	Neck Shear Force lb	Neck Shear Force (N)	Axial Force lb	Axial Force (N)	Head Angle Relative to Torso deg	AIS Rating	Comments
Forward bending 0 deg	43	65.0	(88.2)	177	(787)	-	-	70	1	Pain but no injury
Rearward bending 180 deg	45	22.5	(30.5)	52	(231)	56	(249)	68	0	No injury
Lateral bending 90 deg	45	33.3	(45.2)	178	(792)	-	-	43	0	No injury
Lateral bending 135 deg	45	13.3	(18.0)	70	(311)	80	(356)	-	0	No injury
Lateral bending 45 deg	45	23.0	(31.2)	99	(440)	37	(165)	-	0	No injury

NOTE—These values are not necessarily upper bounds of tolerance load reactions between the head and the neck at the occipital confyles. They are all tolerable loads.

TABLE 7—NECK REACTIONS CALCULATED AT THE OCCIPITAL CONDYLES FOR DYNAMIC HUMAN CADAVER TESTS

Loading Configuration	Ref.	Neck Bending Moment ft-lb	Neck Bending Moment (N·m)	Neck Shear Force lb	Neck Shear Force (N)	Axial Force	Head Angle Rrelative to Torso deg	AIS Rating	Comments
Forward bending 0 deg	43	140	(190)	357	(1588)	—	88	0	No damage
	43	130	(176)	437	(1944)	—	69	0	No damage
Rearward bending 180 deg	36	35	(47)	—	—	—	—	0	No damage
	36	42	(57)	—	—	—	—	3	Ligamentous damage

5.6 Neck Injury Due to Head Loading—The neck can be injured by loading of the head. During head loading, some or all of the head load is transmitted to the torso by the neck structure. The magnitude of the transmitted load is dependent on the location and direction of the head load, the inertia of the head, and the configuration of the cervical spine when the head load is applied. For example, if the neck is straight when a fore/aft or lateral head load is applied, then the neck may undergo significant bending prior to transmitting large neck loads to the torso. However, if a load is applied to the head colinear with the cervical spine and the neck is straight, large tensile or compressive neck loads may be transmitted to the torso with little neck distortion.

Hodgson and Thomas (Ref. 49) discussed the effect of neck configuration on the magnitude of axial compressive loads transmitted by the neck, and the location and type of neck injuries for impacts to the top of the head. They measured bone strains on the anterior surfaces of the third, fifth and seventh cervical vertebrae of human cadavers for various neck articulations. For a given applied head load, the anterior cervical body strains were the lowest when the vertebrae were aligned; i.e., neck straight. This implies that the neck behaved as a column and that the neck compressive load should be a good indicator of the potential for neck injury. When the neck is flexed, the cervical vertebrae are subjected to a combined axial compression and bending moment. For this condition, the axial compression load alone may not be a good indicator of the potential for neck injury.

Culver, et al (Ref. 50), subjected human cadavers to superior-inferior head impacts. The necks of the cadavers were not flexed and a padded impactor was used to preclude skull fractures. A summary of the peak applied head load and observed neck trauma is given in Table 8. The mean axial compressive peak head load producing neck trauma was 1620 lb (7.22 kN), and the range was 1060 lb (4.71 kN) to 1990 lb (8.85 kN).

Crown impact tests were conducted by Nusholtz, et al (Ref. 51). In these tests, the thickness of the padding covering the impactor surface was varied to give different force-time characteristics. A summary of the peak applied head loads and resulting neck damage is given in Table 9. The peak head loads which produced neck damage had a range of 405 lb (1.8 kN) to 2495 lb (11.1 kN) and a mean value of 1210 lb (5.4 kN). The authors concluded that the initial configuration of the cervical spine had a major influence on the load carrying capacity and damage patterns which were observed. Note that both Culver, et al., and Nusholtz, et al., measured applied HEAD loads. The axial compressive NECK loads corresponding to these applied HEAD loads could be smaller due to head mass inertial effects.

TABLE 8—COMPRESSIVE HEAD LOADS AND RESULTING NECK DAMAGE DESCRIPTIONS FOR SUPERIOR-INFERIOR HEAD IMPACTS TO CADAVERS WITH NECKS STRAIGHT (50)

Axial Compressive Head Load lb	Axial Compressive Head Load N	Neck Injury Description
1490	6620	No Fractures
1510	6700	No Fractures
1560	6950	No Fractures
1060	4710	Spinous processes of C4, C5, C6 fractured. Transverse process of C5 fractured. Body of C5 crushed on right side.
1360	6050	Tips of spinous processes of C3, C4, C5 fractured.
1580	7030	Spinous processes of C7 and T1 and both transverse processes of T1 fractured. Right transverse process of C7 crushed.
1620	7200	Body of C5 fractured.
1680	7450	C5–6 disk crushed. Spinous process of C2 fractured from body at arches. Tip of spinous process of C6 fractured.
1800	8000	Spinous processes of C1, T1, T2 fractured through arches. Tips of spinous processes of C2, C4, C7 fractured.
1900	8450	Complete fracture from body of C3 and C4 left transverse processes. Chip fractures of spinous processes of C5, C6, C7, T2.
1990	8850	C3–4, C4–5, C5–6 disks crushed. Transverse processes of C5 and T1 fractured, body of T2 severely crushed.

TABLE 9—COMPRESSIVE HEAD LOADS AND RESULTING NECK DAMAGED DESCRIPTIONS FOR SUPERIOR-INFERIOR HEAD IMPACTS TO CADAVERS WITH VARIOUS HEAD-NECK-TORSO ANGLES (51)

Axial Compressive Head Load lb	Axial Compressive Head Load N	Neck Damage Description
405	1800	Fracture of C6 spinous process. Fracture of C7 lamina, articular process and body. Rupture of anterior and posterior longitudinal lagaments and ligamentum flava between C6–C7 with disk involvement.
495	2200	Extension/compression type damage. (osteoporotic)
515	2300	Fractures of C5 and C6 bodies. Fracture of C5 spinous process. Fracture of C6 lamina. Rupture of anterior longitudinal ligament between C5–C6 with disc involvement.
740	3300	Fractures of C3–C7 spinous processes. Fracture of C2–C3 disc with displacement. Rupture of anterior longitudinal ligaments between C3–C4 and C5–C6 with disc involvement. Rupture of C2 posterior longitudinal ligament.
740	3300	Fractures of C3–C4 spinous processes. Fracture of C4 articular capsule. Rupture of longitudinal ligament between C3–C4 with disc involvement.
1280	5700	Fracture of C4 spinous process. Rupture of anterior longitudinal ligament between C2–C3 with disc involvement.
1350	6000	Fractures of C5 and C7 bodies. Rupture of anterior longitudinal ligament at C5. Fracture T2 body.
1395	6200	Fractures of C7 and T1 spinous processes. Rupture of anterior longitudinal ligament between C6–C7 with disc involvement.
1595	7100	Bilateral joint laxity between C4–C5. Fracture of T4 body. Rupture of inter - and supraspinous ligaments between T3–T4.
2315	10300	Rupture of supra - and inter spinous ligaments and ligamentum flavum between C7–T1 with disc involvement. Fracture of T1 body.
2495	11100	Rupture of anterior longitudinal ligament between C3–C4 with disc involvement. Fracture of T3 body.
630	2800	No damage

In an accident reconstruction program, Mertz et al. (Ref. 52), exposed an instrumented Hybrid III dummy to head impacts using a spring propelled, tackling dummy which has produced serious neck injury to football players. In these tests, the dummy was oriented so that the load was applied to the top of the head, loading the neck structure in compression with minimal head rotation. This configuration was chosen to produce the maximum value of neck compression force for the impact velocity used. The neck compressive load measured by the Hybrid III dummy should be representative of the upper bound of the maximum axial compressive load that an equivalent weight human would experience for the same impact velocity since the relatively soft stiffness of the tackling dummy bag should mask the effect of the relatively stiff neck response required to obtain a maximum compressive loading. Based on their results, they proposed a time-dependent, injury criterion for axial compressive neck loads, Figure 2. Exceeding the criterion implies that major neck injury (permanent impairment of a body function) is likely. However, being below the criterion does NOT imply that major neck injury will not occur if other neck loading modes are present.

5.7 Thorax—The human thorax (or chest) is a ribbed shell (rib cage) containing the following important organs: heart, lungs, trachea, esophagus, and major blood vessels. The size and shape of the thorax depends on the age and sex of the individual, but roughly it may be described as a truncated cone with its depth less than its breadth. The rib cage is a semi-rigid structure which provides protection to the internal organs and facilitates the mechanics of respiration.

5.7.1 THORACIC INJURIES—Thoracic injuries may be divided into two types: (a) injuries to the internal thoracic organs and (b) injuries to the rib cage. Injuries to the internal organs include pneumothorax[3], hemothorax [4], ruptures of the heart, ruptures of the arteries connected to the heart, injury to the cardiac muscle, lung contusion, bruising, or rupture. Of these, the most frequent and most serious is the rupture of the thoracic aorta which is the major artery attached to the heart. Cardiac injuries are thought to be caused by compression of the heart between the spinal column and the sternum (breast bone). There is an increased possibility of cardiac rupture if the heart is in that portion of its pumping cycle where it is full of blood. Tears of the aorta usually occur just beyond the aortic arch at its junction with the subclavian artery. The tears are usually transverse to the vessel axis. The exact mechanism of failure is not yet understood. Injuries to the rib cage include fractures of the ribs and sternum, and less often, dislocations and fractures of the thoracic vertebrae. Rib fractures become dangerous if the broken rib ends are displaced to the point where they can puncture internal organs or are numerous enough to inhibit adequate inspiration.

3. These denote, respectively, free air blood in the sac surrounding the lung tissue
4. These denote, respectively, free air blood in the sac surrounding the lung tissue

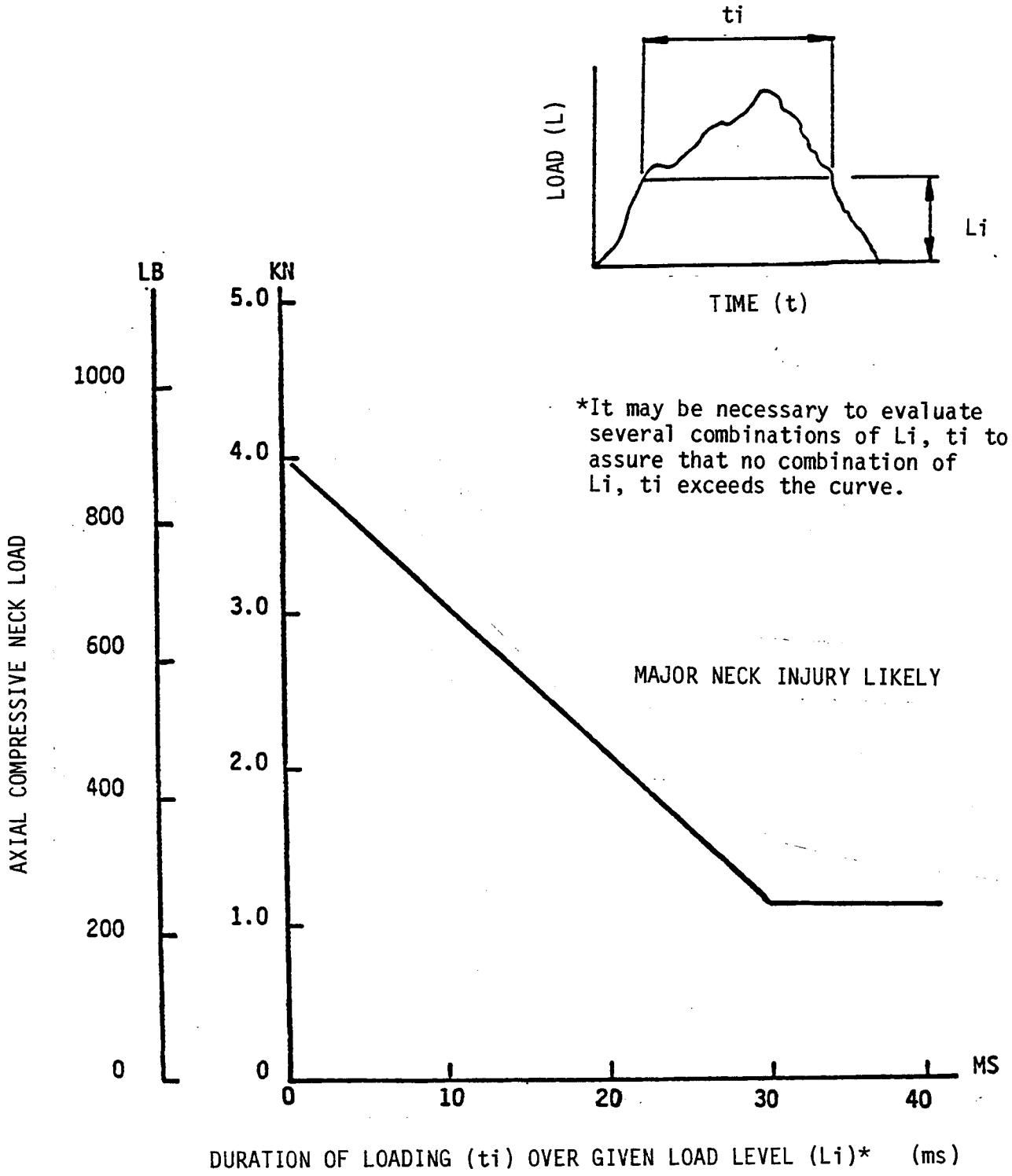

FIGURE 2—TIME DEPENDENT INJURY CRITERION FOR AXIAL COMPRESSIVE NECK LOADS, NECK STRAIGHT (REF. 52)

5.7.2 THORACIC INJURY CRITERIA—Several parameters have been suggested for monitoring the effect of a blow to the thorax based on thoracic acceleration, force, deflection, or some combination of these. Chest impact studies have been conducted by a number of researchers using both embalmed and unembalmed cadavers, experimental animals (monkeys, dogs, and pigs) and, for quasistatic chest loading, volunteers. There are difficulties associated with the use of any of the above test subjects in determining thoracic injury tolerance. With cadavers, corrections may be needed to account for their lack of muscle tone and lung inflation as compared to living subjects. Rib fractures are a commonly employed measure of thoracic injury with cadavers but this factor is highly age dependent. Data obtained with tests on animals must be scaled to account for size and shape differences of their thoracic cage as compared to the human; injury interpretation is complicated by anatomical differences between the human thoracic organs and those found in experimental animals.

The majority of experimental chest impact studies have involved frontal impacts employing either simple impactors or belt restraint systems. A lesser amount of data are available on lateral chest impacts. There are no data available at this time on oblique impacts to the chest.

5.7.2.1 *Chest Deflection*—Researchers have generally concluded that chest deflection is a response measure which shows good correlation with chest injury produced by blunt frontal impacts. Neathery, et al (Ref. 53) has been a major advocate of this approach. He analyzed test results on 24 cadavers that had received frontal blunt thoracic impacts delivered by a simple impactor. Employing regression analysis, Neathery related their chest traumas (using the 1971 AIS scale) to their chest deflection (normalized by chest depth) and ages at death.

Based on an injury level of AIS 3 (severe; not life-threatening), and a median driving age of 45 years old, Neathery recommended the following sternal deflection limits:

TABLE 10—

Occupant Size	Recommended Sternal Deflection Limit for AIS 3 mm	Recommended Sternal Deflection Limit for AIS 3 in
5th percentile female	60	2.36
50th percentile male	75	2.95
95th percentile male	90	3.54

The preceding recommendation for the 50th percentile male is consistent with observations made previously by Melvin, et al (Ref. 54). Melvin proposed a chest deflection limit of 1.75 in (44 mm) if rib fracture was to be avoided; he also concluded that a deflection range of 2.5–3.0 in (64–76 mm) would correspond to an AIS injury level of 3.

In a subsequent re-analysis of the data used by Neathery, Viano (Ref. 55) emphasized the distinction between skeletal and non-skeletal thoracic injuries. By separating these injury classes, Viano noted that internal injuries (which can be life-threatening) only began to appear at P/D ratios[5] of approximately 0.40. At this deflection level, the rib cage has lost its structural integrity due to multiple rib fractures. Viano's P/D limit of 0.40 is slightly greater than Neathery's recommended limit of 0.387; however, Viano's limit represents the onset of life-threatening injuries, (AIS = 4) whereas Neathery's limit represents an AIS level of 3 which is serious, but not life-threatening. It should also be noted that Viano's analysis did not correct for the effects of age levels as did Neathery's.

5. P/D = penetration divided by pre-impact chest depth

The primary disadvantage of the deflection criterion is the difficulty of performing the measurement, both on biological specimens as well as on test devices. A further complication is that a single deflection measurement is not generally representative of the complete thorax deformation behavior, unless the nature and location of the impact is well understood beforehand and the transducer positioned accordingly.

5.7.2.2 *Chest Acceleration*—The practical difficulties of the deflection criterion have led many researchers to conclude that acceleration measurements offer an attractive alternative. Stapp (Ref. 56) reports on numerous tests where volunteers were subjected to decelerative restraint environments. For a series of "rocket sled" tests where the volunteers were restrained by air force restraint harnesses, cardiovascular shock (drastic drop in blood pressure immediately post test) was noted in several tests where the peak sled deceleration ranged from 26–38.5 G with deceleration onsets of 896–1373 G/s. Unfortunately, these subjects were not instrumented with chest accelerometers.

Mertz and Gadd (Ref. 57) report that an instrumented stunt man experienced chest accelerations of 46 G's while impacting a thick foam mattress with his back after diving 57 ft (17.4 m) from a tower. Viano, et al. (Ref. 58), measured the chest acceleration of a performer who routinely dove from a height of 34.5 ft (10.5 m) into a shallow pool, impacting the water's surface with his belly. They measured thoracic spine and sternal accelerations of 25 G and 224 G, respectively, for a 15 ft (4.6 m) dive. The authors extrapolated these measured results to 68 G and 380 G, respectively, for his normal performance height.

FMVSS 208 currently specifies as acceptable any acceleration pulse which "... shall not exceed 60 G except for intervals whose cumulative duration is not more than 3 ms". Previously, MVSS 208 had applied a Severity Index to the chest acceleration pulse. This index was calculated in exactly the same manner as the head Severity Index discussed previously, and the limit of 1000 was the same as that for the head. Both the 60 G and the chest Severity Index limits are based on the resultant acceleration measured at the center of gravity of the dummy thorax.

Accelerations measured at a single point (as described above) cannot adequately represent the complete response of the thorax. For this reason, Robbins, et al (Ref. 59) employed a sophisticated approach to accelerometer usage for determining the overall response of the thorax. His group performed tests on animals and cadavers instrumented with ten accelerometers mounted at eight different locations on their rib cages and backbones. Test conditions included frontal and, subsequently, lateral impacts (Ref. 60). The instrumentation, which was later increased to twelve accelerometers, was chosen to be consistent with dummy instrumentation usage. Their intent is to determine a predictive function which will enable these accelerometer signals to be combined in a manner that is related to the thoracic injury. This approach requires a large number of tests and the use of a computer to generate regression models.

5.7.2.3 *Shoulder Belt Load*—In an analysis of data from 108 frontal tests with seat belted cadavers conducted at five research institutes, Eppinger (Ref. 61) formulated an equation which predicts the number of observed thoracic fractures (this includes fracture of the ribs, sternum, and clavicles) based on the maximum upper torso belt force, the cadaver weight, and the cadaver age at death. As an example of the application of this method, Eppinger chose to use the age and weight distributions of the U.S. automotive fatality population in a particular 30 mph (13.4 m/s) frontal crash with a particular belt restraint system. From this he determined that the total number of rib fractures to the target population would be minimized if shoulder belt forces could be limited to 1300–1500 lb. Eppinger employed a 12 in (305 m) stroke limit on his belt system which precluded a lower optimal force level.

Foret-Bruno, et al (Ref. 62), reported on the relationship between vehicle occupant thoracic injuries and shoulder belt loads estimated from frontal accidents in which the occupant was restrained by a unique energy absorbing lap/shoulder belt system which allowed the shoulder belt load to be approximated. No chest injuries were received by occupants less than 30 years old for shoulder belt loads under 1650 lbs. (7.30 kN). Beyond age 50, fractures began to occur at about 950 lbs. (4.20 kN) of belt load. The authors compared these results to Eppinger's analysis and concluded that cadavers could be expected to sustain three to five more rib fractures than the living crash victim under similar impact conditions.

The disadvantage of employing shoulder belt load as an injury criterion lies in its sensitivity to shoulder belt geometry. The force on the torso is not only a function of the belt loads, but also is dependent on the angles of the belts relative to the torso. These belt angles can be expected to vary among belt restraint systems since they are a function of such variables as anchorage locations, seat height, seat stiffness, and webbing properties. Belt angles can also be expected to change with the occupant's movement during the impact event.

5.7.2.4 *Lateral Loading*—Some recommended limits for side impact to the chest come from an animal and cadaver study by Stalnaker (Ref. 63). Two impact surfaces were considered in this investigation. A flat 6-in (15.2 cm) diameter surface was employed for blunt impacts and a simulated armrest was employed for concentrated impacts. Stalnaker concluded that a lateral chest deflection of 31 percent of chest width produced by the blunt surface would result in an AIS injury level of 3; the comparable non-fracture limit was found to be 22 percent.

Tarriere, et al. (Ref. 64), investigated the tolerance of the thorax to lateral impacts by dropping unembalmed cadavers, suspended horizontally, against a broad, flat load cell surface. Rigid and padded conditions were employed. The cadaver thorax instrumentation included triaxial accelerometers and a deflection rod installed transversely through the rib cage and viewed photographically. The predominant traumfa was rib fractures with no visceral lesions being found. Mineralization tests were conducted on rib samples, post-test, to determine the suitability of the specimens employed. Force, acceleration and deflection were all considered as possible injury criteria, but deflection was found to provide the best correlation to trauma severity. A thorax relative deflection of 30 percent was found to equate to an injury level of AIS ≤ 3. This value compares reasonably well with Stalnaker's recommendation.

An important consideration in conducting side impact vehicle tests is the placement of the upper arm of the surrogate. The upper arm can be placed alongside the chest or raised, thereby exposing the thorax to direct impact. To resolve this question, it would be necessary to know the circumstances of arm placement in field accidents as well as the biomechanical effects of arm positioning on subsequent injury patterns to the thorax and arm.

Cesari, et al. (Ref. 65), studied the latter issue by conducting lateral impact tests to eight unembalmed cadavers. In most of these tests, the arm was alongside the thorax so that the thorax was impacted through the arm. These results were compared to those of a similar series, conducted previously, in which the arm was raised and the thorax was struck directly. Their impactor was a spherical sector with a 23.6 in (60 cm) spherical radius and a 6.9 in (175 mm) sector radius and weighed 51 lb (227 N). Impact velocities ranged from 6.2–16.8 mph (10–27.1 km/h). The arm was found to provide some protective value to the thorax when the arm received the blow. This protective effect was generally equivalent to a 10 percent change in impactor velocity. The nature of the thoracic injuries (as distinct from their severity) was not appreciably changed by the presence of the arm. Rib fractures were the most prevalent trauma but intrathoracic injuries also occurred. There was only one arm fracture, on a relatively severe test.

5.8 **Abdomen—**The abdomen is the least understood region of the body from the load tolerance viewpoint. It contains a variety of organs which can be exposed to impact forces. The organs most frequently injured as a result of blunt abdominal trauma are the liver, kidneys, spleen, intestines, pancreas and the urinary bladder. Only the liver and the spleen are partially protected by the lower aspects of the rib cage. Diagnosis and localization of organ injury in the abdomen are difficult, and the serious threats of hemorrhage and infection require prompt surgical intervention when these organ injuries are present.

5.8.1 TOLERANCE OF ABDOMINAL ORGANS (FRONTAL IMPACTS)—A large body of clinical literature has evolved over the years to document the various forms of injuries produced by blunt abdominal trauma. In contrast, there are only a few studies available on the loading condition, force levels, and impact velocities that characterize typical accident situations. One of the earliest of these is a 1953 report by Windquist, Stumm, and Hansen (Ref. 66). They employed upright seated, forward facing hogs to examine the effect of abdominal impacts against restraining belts (improperly worn lap belts) as well as objects that might be struck in an aircraft

cockpit. These objects were a control wheel, a stick-like protrusion[6] and a large, flat surface similar to a radio box. The animals received impacts in both their midriff and lower abdominal regions at velocities of 20 and 40 ft/s (6.1 and 12.2m/s). All of the high velocity exposures (with belt loop forces ranging from 2360–6660 lb (10.5–29.6 kN)) were fatal. A force of 1080 lb (4.80 kN) against the 10 in (254 mm) square radio surface, a force of 893 lb (3.97 kN) against the projecting peg, and a 750 lb (3.34 kN) loop force through the abdominal belt were all considered survivable. Complete results are available in Table 11 which was given in a paper by Mertz and Kroell (Ref. 67).

A later study by Stalnaker, et al, probably provides the most extensive test results yet published on abdominal impact (Ref. 68). A series of 96 abdominal tests were carried out on four animal species-Rhesus monkey, Squirrel monkey, baboon, and pig. Various sized impactors were employed to simulate common automotive injuries. The abdomen was divided into three zones (upper, middle, and lower) which were analyzed separately. The voluminous data generated in this project was submitted to computer assisted statistical analysis to obtain correlations between the various impact parameters and the estimated injury severity ratings which were obtained on autopsy. Their overall results are summarized in Figure 3.

5.8.2 TOLERANCE OF ABDOMINAL ORGANS (LATERAL IMPACTS)—One of the earliest studies on lateral tolerance of the abdomen was reported by Stalnaker, et al. (Ref. 32), in 1973. These researchers impacted a variety of live, anesthetized primates in the right and left sides of their abdomens, employing a scaled armrest on a 22 lb (98 N) moving striker. The force levels required to produce injury varied significantly with the site of the impact. The upper portion of the abdomen was found to be more easily injured than the lower portion of the abdomen.

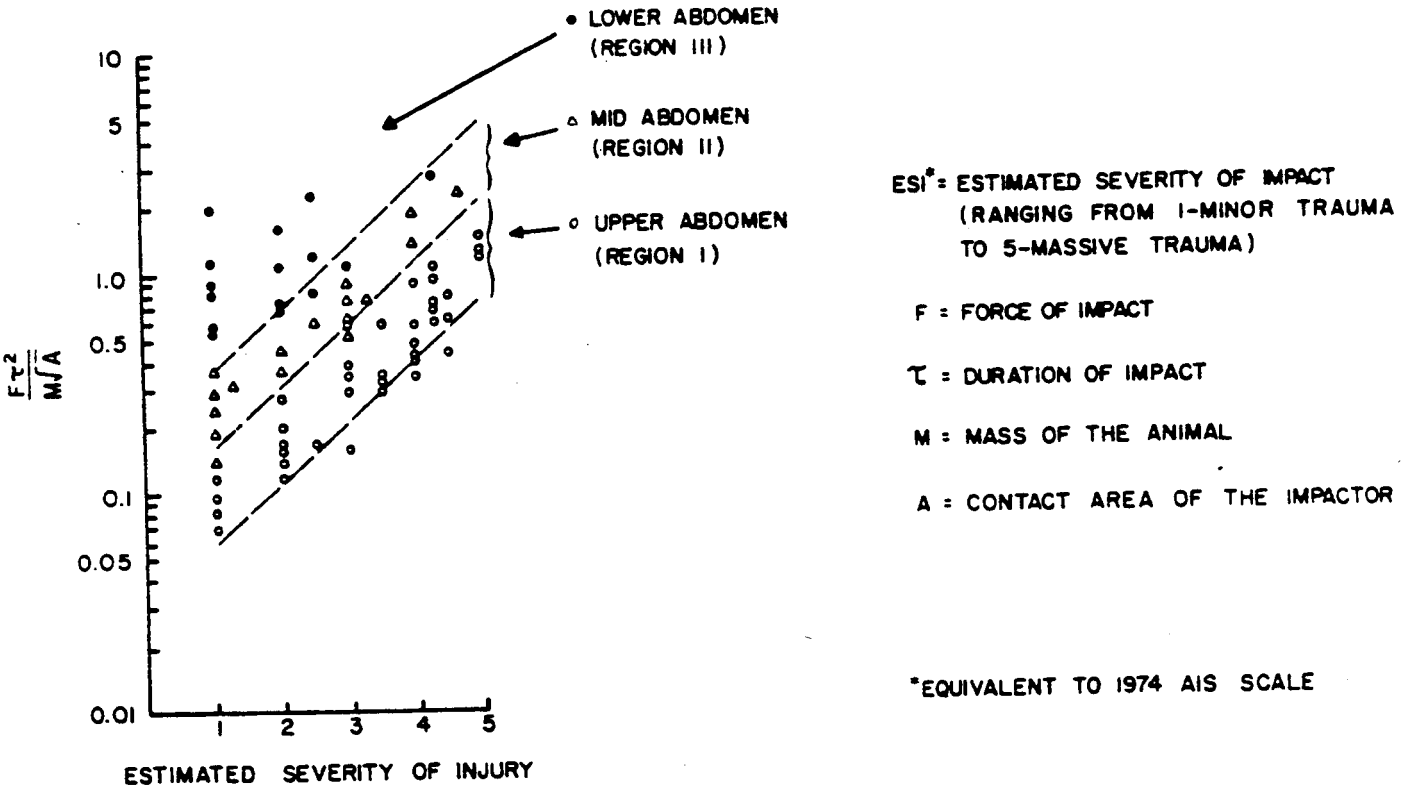

FIGURE 3—EXPERIMENTAL SCALING FACTOR FOR ABDOMINAL INJURY SENSITIVITY

6. struck end-on

TABLE 11—MIDRIFF LOADING (WINDQUIST, ET AL.)

Type of Exposure	Hog Weight lb	Hog Weight N	Impacct Velocity ft/s	Impact Velocity (m/s)	Maximum Force Applied lb	Maximum Force Applied kN	MFRT[1] PD[2] ms/ms	Approx. Body Compression % of Normal Thickness	Descrption of Injuries
Control Wheel Impingement Block	95	422	20.7	(6.3)				40	**Survivable**-Small subpleural and subendocardial hemorrhages; incomplete defect in anterior wall of stomach.
	100	445	40.7	(12.4)				80	**Fatal**-Massive internal injuries including bilat. fract disloc's at costochondral junct., ruptured diaphragmatic hernia, large inguinal hernias, perforation of stomach and intestines and laceration of spleen.
	166.5	740	39.1	(11.9)					**Fatal**-Multiple comp. rib fract. bilaterally; destruction of liver; lacerations of pericardium, heart, right lung, diaphragm, colon, and peritoneum.
Ten Inch Square Impingement Block	104	463	20.3	(6.2)	1080	(4.8)	48/95	30	**Survivable**-Small subpleural and subendocarial hemorrhages; subserosal hemorrhage of the colon.
Ten Inch Square Impingement Block	95	422	39.5	(12.0)	2360	(10.5)	22/85		**Fatal**-Massive internal injuries including multiple rib fract, and disarticulartion, lacerations of pericardial sac, heart, pulmonary artery, stomach, and intestines.
	156.5	696	40.3	(12.3)	6660	(29.6)	38/72		**Fatal**-Massive internal injuries including multiple rib fract., hemorrhage and emphysema of lungs, multiple lacerations of liver and spleen, multiple ruptures of colon.
Projecting Peg Impingement Block	175	779	17	(5.2)	893	(4.0)	68/135	70	**Survivable**-Single rib fracture.
	149	663	39.7	(12.1)	3085	(13.7)	27/95	90	**Fatal**-Massive internal injuries including puncture into right pleural cavity, comp. rib fract. bilaterally, laceration of pericardium, heart, right lung, diaphragm, liver, intestines, stomach, and peritoneum.
Control Wheel Impingement Block	185	823	23.6	(7.2)	2365	(10.5)	68/100	80	**Fatal**-Petechiae over lungs, subepicardial ecchymosis over anterior portion of interventricular septum, hemorrhage in diaphragm, subcapsular hemorrhage of liver, multiple ruptures of wall and colon.
	187	832	39.6	(12.1)	5080	(22.6)	46/88	80	**Fatal**-Massive internal injuries including laceration of rectus abdominus muscle, ruptured diaphragmatic hernia, lacerations of liver and spleen, pericapsular hemorrhage about left kidney, ruptures of colon.
Three Inch Wide Abdominal Belt	62.8	279	19.3	(5.9)	750 (loop load)	(3.3)	28/52		**Survivable**-Subendocardial hemorrhage over septal portion of left ventricle and multiple subserosal hemorrhages of mid portion of jejenum.
	69.8	310	44.2	(13.5)	4700 (loop load)	(20.9)	38/60		**Fatal**-Massive internal injuries including ruptured diaphragmatic hernia, ruptures of stomach and colon, fragmentation of spleen, lacerations of kidneys and liver.

1. Maximum force rise time.
2. Pulse duration.

Walfisch, et al, (Ref. 69), conducted lateral impacts to eleven unembalmed cadavers to determine lateral abdominal tolerance levels. Based on their previous accident study, they concluded that liver injuries were the most common of the serious abdominal traumas. Accordingly they impacted only the right side of their cadavers (which contains the bulk of the liver) in order to obtain results which they felt would be conservative. The cadavers were suspended horizontally, right side downward, and allowed to free fall from a height of 3.3 ft (1 meter) or 6.6 ft (2 meters). The abdomen struck a simulated armrest which was mounted to a load cell. The armrest was 2.8 in (7 cm) wide (corresponding to the s-i direction of the cadaver) and its depth and stiffness in the l-r direction were varied by employing various types of supporting material under a wooden form. Armrest depth ranged from 1.2 in (31 mm) – 2.2 in (55 mm). Abdominal penetration was obtained photographically, but an ambiguity developed in this measurement due to sagging of the abdomen and crushing of some of the armrests. In addition, the abnormal attitude of the cadaver relative to the seated position may affect the positioning of the abdominal organs and hence the damage results. Their force measurements and abdominal (liver) injury ratings are provided in Table 12.

TABLE 12—CADAVER DROP TESTS ON THE ABDOMEN (LATERAL)

Test No.	Height of fall (m)	Protrusion of simulated armrest (mm)	Supporting Material for armrest	F. Max da N	Abdominal AIS
205	1	31	rigid	160	0
206	1	51	rigid	535	4
209	1	51	polystyrene	380	4
210	1	51	polystyrene	415	3
211	1	53	phenespan	170	0
212	1	55	polystyrene	150	(1)
219	1	41	rigid	195	1
213	2	55	polystyrene	490	3
215	2	31	rigid	510	5
216	2	51	rigid	420	1
217	2	41	rigid	500	5

1. diseased livers - subjects eliminated from analysis

5.8.3 LOADING OF THE ABDOMEN BY A LAP BELT—Walfisch, et al. (Ref. 70), subjected fourteen unembalmed cadavers to a series of sled impacts in which the lap belt and cadaver were configured to promote over-riding of the pelvis by the lap belt. Belt loads and abdominal penetrations were measured during the tests and resultant injuries were determined post test. The damage found on these cadavers was considered to be similar to those of accident victims. The nature of the cadaver damage and their frequency of occurrence were:

Fracture of the lumbar spine	4
Tearing of the mesentery	2
Damage to the liver	2
Fracture of the pelvis	1
Perforation of the colon	1

This work led the authors to recommend that a conservative tolerance level for the abdomen (lap belt above pelvis) would be a lap belt load of 450 lb (2.0 kN) per side accompanied by an a-p lap belt intrusion of 1.4 in (35 mm) per side. In a later paper, Leung (Ref. 71), a member of the earlier research team, increased the recommended abdominal tolerance level to an average lap belt tension of 790 lb (3.5 kN), (average of

inboard and outboard lap belt loads) accompanied by an average lap belt penetration of 1.5 in (39 mm) (average of the a-p belt penetrations on the right and left side of the abdomen). It should be noted that their load tolerance recommendation refers to the lap belt load and not to the force on the abdomen. This recommendation should be applied cautiously to routine vehicle testing since lap belt geometries in some vehicles and abdominal stiffnesses of most test devices can vary appreciably from the conditions obtained in the referenced study. In addition, their recommendation may not be applicable to the situation in which lap belt over-ride occurs on only one side of the pelvis since that condition was not investigated.

Research by Nusholtz, et al, (Ref. 72), indicated that extreme care is needed when determining abdominal tolerance levels through the use of post-mortem specimens. This group compared the injuries to live and post-mortem primate subjects produced under blunt impacts delivered laterally to the thoraco-abdominal region. The live primates were injured more seriously than their corresponding post-mortem subjects. They suggested that the disparity may have been due to the lack of pressurization in the post-mortem specimens in this series.

5.8.4 LOADING BEHAVIOR OF ABDOMINAL ORGANS—Melvin, et al., studied the loading behavior of livers and kidneys which were surgically mobilized[7] from anesthetized Rhesus monkeys and then placed on an uniaxial load cell while still being supplied with blood by the living animal (Ref. 73).

Tests were performed at ram speeds of 120, 6000, and 12,000 in/min (0.05, 2.5, and 5.1 m/s) and average stress/strain[8] curves were obtained. In addition, the resulting injury severity was estimated immediately after impact using an ESI[9] injury scale of 1 (minor) to 5 (massive).

The authors concluded that both organs were sensitive to loading rate effects with the liver being more affected. A liver trauma rated as an ESI of 3+ was produced at a dynamic average stress level of approximately 45 psi (310 kPa). The kidney, with its thick, tough capsule, displayed wide variations in injuries at a given dynamic stress level. Its injury severity appeared to be more properly related to its strain level. The organ injuries in this study were noted to be similar to those seen clinically.

5.9 Lower Extremities—The structural elements of the lower extremities consist of the pelvis, the femurs, the tibias, the smaller fibulas, and the ankle and foot bones. In addition, there are the patellas (bony kneecaps) which cover the knee joints in front and serve as termini for ligaments and tendons. Also noteworthy is the pronounced offset of the head of the femur where it articulates in a ball and socket type joint at the pelvis.

5.9.1 TEST TECHNIQUES—Two types of test procedures have been employed to study the strengths of the patella/femur/pelvis bone complex. One technique employs an instrumented moving impactor to load the upper leg of a stationary, seated cadaver. The other procedure employs a moving test sled to propel the entire cadaver against instrumented surfaces arranged to simulate a vehicle interior.

5.9.1.1 Impactor Test Data—Powell, et al. (Ref. 74) tested the legs of nine cadavers and obtained fractures at loads ranging from 1600–2970 lb (avg. 2360 lb) (7.12–13.20 kN) (avg. 10.50 kN). Eighty percent of their legs suffered patellar fracture, 33 percent sustained condylar fractures (the portion of the femur adjacent to the patella), and only 6.7 percent were fractures to the shaft of the femur. They attributed the fracture patterns to the rigid impactor which they used.

7. Surgically separated from its surrounding tissues but leaving connecting blood vessels intact.

8. $$\text{average stress} = \frac{\text{impactor force}}{\text{iimpactor area}}$$

$$\text{average strain} = \frac{\text{deflection}}{\text{initial thickness of specimen}}$$

9. This scale is equivalent to the 1974 AIS scale.

Melvin, et al. (Ref. 75), employed an impactor with 1 in (25 mm) of Ensolite padding to test the femurs of fourteen stationary, seated cadavers. No fractures were obtained below 3000 lb (13.30 kN) and it was noted that a threshold impactor momentum of 40–50 lb·s (180–220 N-s) appeared to be necessary to cause fracture. Their relatively high fracture load levels (as compared to previous studies) were attributed to their exclusive use of unembalmed cadavers. All of these fractures were in the patella or in the distal third and supracondylar region of the femur.

Viano et al. (Ref. 76), conducted a series of axial knee impact tests on a total of six seated cadavers. A 22.3 lb (10.1 kg) impactor was used in conjunction with varying degrees of padding. The skin was removed from the impacted areas, but the structural integrity of the knee joint, including the ligaments, was left intact. This procedure was used in an effort to measure the time of fracture initiation, based on analysis of high-speed movies of the patella/femur during impact.

All six of the tests with rigid impactors produced both patella and femoral shaft fractures, and also produced either a condylar or neck fracture. The peak force ranged from 3010 to 6410 lb (13.4 to 28.5 kN), with an average of 4110 lb (18.3 kN). Many of the specimens, especially those impacted without padding, had multiple fractures. The authors contend that most of these fractures were initiated after the load peaks. The average load that the authors associated with shaft fractures was 2300 lb (10.2 kN) compared to the average peak load of 3350 lb (15.0 kN). The two tests conducted with lightly padded impactors both produced bilateral condylar fractures. The peak loads were 3600 and 3460 lb (16.0 and 15.4 kN). Only two of the five tests with thickly-padded impactors produced fractures (one condylar and one femoral shaft) and both of these involved cadavers which were considered by the authors to have bones in "abnormal" condition. The three "normal" specimens produced peak loads of 1190, 3100 and 3150 lb (5.3, 13.8 and 14.0 kN), and presented no fractures.

A shortcoming of some of these studies with moving impactors has been the poor correspondence in the location of the femur fractures as compared to those found in the field. Melvin, et al. (Ref. 75), described the distribution of lower limb fractures that occur in real world collisions. They found 142 unbelted vehicle occupants who suffered lower limb fractures in frontal collisions. Of these, thirty-nine (27 percent) had patellar or distal femur (adjacent to kneecap) fractures. The impactor studies have produced proportionately more such fractures.

This disparity is felt to be due to the non-representative ridigity and orientation of the laboratory impactors. The rigid or near-rigid impactors produced pulse durations of only 3 to 10 ms, as compared to the 30 to 50 ms durations which characterize actual instrument panel impacts. In addition, the impactors were aligned with the femur, maximizing its compressive load carrying capacity. In accidents, it is more likely that the knee impact force vector and the femoral axis will not be aligned, thereby resulting in greater bending stresses and uneven force distribution between the patella and condyles. These factors reduce the load carrying capacity of the legs. For these reasons, impact tests of 30–50 ms duration, with non-aligned femurs, are probably more relevant to the automotive collision environment than are the results from the various impactor studies.

TABLE 13A—DYNAMIC FRACTURE FORCES FOR FEMUR, PATELLA, AND PELVIS (REF. 77) (RIGHT THIGH/LEFT THIGH)

Right Thigh Cadaver No.	Right Thigh Max Force Applied lb	Right Thigh Max Force Applied kN	Result	Left Thigh Max Force Applied lb	Left Thigh Max Force Applied kN	Result
1	950	4.23	Supracondylar fracture (bone defect suspected)	1400	6.23	intertrochanteric fracture (fractured through bone screw)
2	1500	6.68	Mid-shaft fracture	1600	7.12	No fractures
3	1500	6.68	No fractures	1600	7.12	No fractures
4	1650	7.34	Supracondylar fracture	1650	7.34	Supracondylar fracture
5	2150	9.57	No fractures	2100	9.35	No fractures
6	2250	10.00	No fractures	2250	10.00	Superacondylar fracture
7	1900	8.46	No fractures	1850	8.23	Superacondylar fracture
8	2800	12.46	No fractures	1750	7.79	No fractures
9	3850	17.13	No fractures	2650	11.79	Dislocated intertochanteric fracture
10	2400	10.68	Comminuted fracture of distal third of shaft and intercondylar notch	1800	8.01	No fractures

FEMUR

TABLE 13B—DYNAMIC FRACTURE FORCES FOR FEMUR, PATELLA, AND PELVIS (REF. 77) (RIGHT KNEE/LEFT KNEE)

Cadaver No.	Max Force Applied lb	Max Force Applied kN‘	Result	Max Force Applied lb	Max force Applied kN	Result
1	950	4.23	No fractures	1400	6.23	No fractures
2	1500	6.68	No fractures	1600	7.12	No fractures
3	1200	5.34	No fractures	1600	7.12	Abnormality-but not adequately identified as fracture
4	1650	7.34	No fractures	1650	7.34	No fractures
5	2050	9.12	No fractures	1550	6.90	No fractures (padded)
	1550 1800 1950 2150	6.90 8.01 8.68 9.57	Heavy abrasion and fracture of patella (unpadded)	1500 1800 2000 2100	6.68 8.01 8.90 9.35	Complete fracture of patella, damage to articular cartliage (unpadded)
6	1700 2050 2250	7.57 9.12 10.00	No fractures Comminuted fracture of patella	1950 2000	8.60 8.90	No fractures ominuted fracture) of patella
7	1900	8.46	No fractures	1850	8.23	No fractures
8	2550	11.35	Comminuted fracture of patella	1750	7.79	No fractures
9	3850	17.13	Linear fracture of patella	2650	11.79	No fractures
10	2400	10.68	No fractures	1800	8.01	No fractures

PATELLA

TABLE 13C—DYNAMIC FRACTURE FORCES FOR FEMUR, PATELLA, AND PELVIS (REF. 77) (RIGHT HIP/LEFT HIP)

Right Hip Cadaver No.	Right Hip Max Force Applied lb	Right Hip Max Force Applied kN	Result	Left Hip Max Force Applied lb	Left Hip Max Force Applied kN	Result
1	950	4.23	No fractures	1400	6.23	No fractures
2	1500	6.68	No fractures	1600	7.12	No fractures
3	1200	5.34	No fractures	1600	7.12	Fractures of superior and inferior rami of pubis
4	1650	7.34	No fractures	1650	7.34	No fractures
5	2150	9.57	No fractures	2100	9.35	No fractures
6	2250	10.00	No fractures	2250	10.00	No fractures
7	1900	8.46	Severe multiple fractures	1850	8.23	No fractures
8	1400	6.23	Possible mid fracture of ischium	1750	7.79	No fractures
	2250	6.90	Severe multiple fractures			
9	2750	12.24	No fractures	1950	8.68	Possible mild fracture of transverse ramus
	3850	17.13	Severe multiple fractures	26.50	11.79	Severe multiple fractures
10	2400	10.68	No fractures	1800	8.01	No fractures

PELVIS

5.9.1.2 Sled Test Data

5.9.1.2.1 Loading Through the Knee Joint—The earliest lower limb studies of automotive interest were conducted by Patrick, Kroell, and Mertz (Ref. 77). Their objective was to determine the strength of the patella/femur/pelvis complex in impacts simulating knees striking instrument panels. Ten embalmed cadavers were tested in full-scale impact sled experiments. The seated cadavers translated forward during sled deceleration to impact against four padded load cells. The head, chest, and each knee struck a separate load cell. These cells were geometrically arranged to simulate the forward surfaces of an automobile passenger compartment. These researchers concluded that the femur was slightly more vulnerable to fracture than the patella or the pelvis, but that distinction was too small to allow confident prediction as to which bone structure would fail first. Their complete results are available in Table 13. A later study by the same investigators obtained loads of 1470, 1710, 1950, and 1970 lb (6.54, 7.61, 8.68 and 8.76 kN) on two cadavers without fractures (Ref. 78).

Viano and Culver (Ref. 79) conducted sled tests with cadavers restrained by a shoulder-belt-plus-knee-bolster configuration (i.e., no lap belt). For six of these subjects the bolster was positioned so that the knees impacted it squarely. No injuries were produced for bolster loads which ranged from 1190 to 1800 lb (5.3 to 8.0 kN) per leg with an average of 1420 lb (6.3 kN).

5.9.1.2.2 Loading Below and Across the Knee Joint—In the preceding studies, the loading of the femur was primarily through the patella and/or femoral condyles and resulted in patella, femur and/or pelvis fractures. If the loading is applied below or across the knee joint, damage to the knee ligaments and/or fractures of the tibia and fibula may result. Viano, et al. (Ref. 80), impacted seated cadavers on the anterior portion of the tibia, just below the knee joint. They found that impactor forces ranging from 740 to 1550 lb (3.28 to 6.89 kN) with an average of 1140 lb (5.09 kN) produced knee ligament tearing and/or tibia-fibula fractures. In two tests, no damage was observed for peak loads of 1090 lb (4.87 kN) and 1290 lb (5.74 kN). For eight impacts which spanned the knee joint (involving both the patella and tibia), knee joint damage was produced for impactor forces ranging from 1330 to 1880 lb (5.91 to 8.36 kN) with an average of 1580 lb (7.02 kN). The predominant injury mode was avulsion of the posterior cruciate ligament from the tibial plateau.

Sled tests were conducted by Viano and Culver (Ref. 79) in which shoulder belted cadavers (without lap belts) impacted knee bolsters. The two below-the-knee leg impacts produced significant ligament tears at peak bolster loads of 790 lb (3.5 kN) and 940 lb (4.2 kN) per leg.

5.9.1.3 Static Tests of Knee Joints—Viano, et al. (Ref. 80), also performed low-speed ligament tolerance tests on five isolated knee joints mounted in a universal testing machine. In these tests, the knee joint angle was maintained at ninety degrees while the tibia was displaced rearward relative to the femur until complete joint failure occurred. Loads corresponding to the initiation of joint failure ranged from 320 lb (1.43 kN) to 575 lb (2.56 kN) with an average of 455 lb (2.02 kN). The corresponding displacement of the tibia relative to the femur at the initiation of joint failure ranged from 0.37 in (9.5 mm) to 1.18 in (30.0 mm) with an average of 0.57 in (14.4 mm). The load corresponding to complete joint failure ranged from 375 lb (1.67 kN) to 675 lb (3.0 kN) with an average of 560 lb (2.48 kN).

5.9.2 THEORETICAL ANALYSES AND PROPOSED INJURY CRITERIA—Viano and Khalil (Ref. 81) have analyzed the stress distribution in the axially-loaded femur. They concluded that the location and magnitude of peak femur stresses can be significantly affected by small shifts in the location of the applied load, such as moving its point of application from one condyle to the other.

Time dependent, compressive force femur injury criteria have been proposed by King, et al. (Ref. 82), and Viano (Ref. 83).

The criterion proposed by King, et al., is:

$$F = A - B \log_{10} T \quad \text{(Eq. 3)}$$

where

F = permissible peak compressive femur force
A = 1370 lb (6.09 kN)
B = 215 lb (960 N)
T = pulse duration in seconds

Viano's proposed criterion is:

for T less than 20 ms

$$F = A - BT \quad \text{(Eq. 4)}$$

and for T greater than 20 ms

$$F = C \quad \text{(Eq. 5)}$$

where

F = permissible peak compressive femur force
A = 5200 lb (23.1 kN)
B = 160 lb (710 N)
C = 2000 lb (8.9 kN)
T = pulse duration in milliseconds

The femur limit currently specified in MVSS 208 is a compressive load of 2250 lb (10 kN) for each femur (Ref. 84). Previous MVSS 208 specifications were 1400 lb (6.23 kN) and 1700 lb (7.55 kN).

5.9.3 CONCENTRATED LOADING OF THE PATELLA—It was mentioned previously (see 4.3.1) that the unique construction of the patella makes it vulnerable to concentrated loadings. This phenomenon was studied by Melvin, et al. (Ref. 85), employing three different impactor sizes, all unpadded. Two of the impactors were flat surfaced, circular areas with diameters of 0.61 in (15.5 mm) and 0.43 in (10.9 mm), while the third impactor was ring shaped with an outer diameter of 0.50 in (12.7 mm) and an inner diameter of 0.25 in (6.4 mm). Ninety embalmed patellas were tested with the results shown in Table 14.

TABLE 14—PATELLA LOCALIZED LOADING (RESULTS AVERAGED OVER ALL TEST SPEEDS)

Impactor Size	Area in^2	Area cm^2	Average Failure Load lb	Average Failure Load kN	Minimum Failure Load lb	Minimum failure Load kN
0.43 in (10.9 mm) dia Circular Impactor	0.15	(0.97)	1030	(4.58)	560	(2.49)
0.61 in (15.5 mm) dia Circular Impactor	0.29	(1.87)	1260	(5.60)	700	(3.11)
0.50 in (12.7 mm) dia Ring Impactor	0.15	(0.97)	1320	(5.87)	650	(2.89)

These tests were carried out at three different test conditions to determine the effect of velocity: -static, 10 mph (4 1/2 m/s) and 20 mph (9 m/s). The patella damage pattern varied dramatically with speed. The impactors caused a clean punch-through of the patella during the static tests but multiple fractures or near total destruction of the patella during the 10 and 20 mph impacts (4 1/2 and 9 m/s). The change in fracture

load levels with speed was mixed. All three impactors displayed little or no variation in average fracture loads as the test speed increased from static to 10 mph (4 1/2 m/s); however, the average fracture load increased by 68 percent for the ring impactor when the speed was increased from 10–20 mph (4 1/2–9 m/s) while the increases for the 0.43 in (10.9 mm) and 0.61 in (15.5 mm) diameter impactors were 13 percent and 14 percent respectively.

5.9.4 PEDESTRIAN INJURY—Pedestrian impacts comprise a different class of lower limb injury since they involve transverse loading of the legs rather than the axial loading discussed previously.

There are several studies dealing with the fracture tolerance of the tibia/fibula to transverse loading. Patrick and Mertz (Ref. 86), quoting unpublished papers of Young, indicated that a concentrated force of 1000–1500 lb (4.45–6.67 kN) fractured a sample of tibias when the force was applied at their distal third (i.e., at one-third of the ankle-knee distance up from the ankle). Kramer, et al. (Ref. 87), performed 209 transverse impacts against the lower legs of cadavers. Their 5.7 in (145 mm) diameter impact cylinder produced a 50 percent frequency of fracture at a force of 970 lb (4.31 kN); their 8.5 in (216 mm) diameter cylinder produced a 50 percent frequency of fracture at a force of 740 lb (3.29 kN).

Pritz, et al. (Ref. 88,89), impacted cadavers which were positioned to simulate a pedestrian stance with most of their weight on the impacted leg. In one series of tests (Ref. 88), fifteen cadavers were impacted laterally with two impact surfaces that simulated the hood edge and bumper of a car. In a second test series (Ref. 89), eleven cadavers were impacted with actual vehicles (two frontal, nine lateral). Knee-thigh-hip injuries were found to occur directly at the impact sites where contact with the vehicle occurred. No pelvis damage occurred when the peak pelvis acceleration was less than 45 G, and AIS = 2 pelvic injuries (Ref. 90) were associated with contact forces of 740 lb (3.3 kN) and 1500 lb (6.7 kN). The bumper force required to produce leg/knee damage was highly variable. This was attributed to inconsistencies in the foot-to-ground contact forces. An analysis of the influences of this effect is given by Bacon and Wilson (Ref. 91). Additional analysis of the Pritz, et al., data (Ref. 88) is given in a paper by Eppinger and Kulkarni (Ref. 92). A major conclusion of the Pritz, et al., study is that primary emphasis should be placed on preventing injury to the knee and hip joints rather than preventing long bone shaft fractures, since joint injuries are more likely to result in some degree of permanent functional impairment (Ref. 93).

6. Limitations of Section 4 Data for Automotive Test Use

6.1 Requirements of an Occupant Simulator—The preceding tolerance data would normally be applied to automotive safety problems by employing an occupant surrogate in an impact simulation to produce a measure of the impact loads that would be imposed on its human counterpart. Such a simulator must be human-like in both its relevant kinematic and impact characteristics in order to perform properly as a human surrogate. Kinematic behavior refers here to the surrogate trajectories during simulated collisions. Impact behavior refers here to the manner in which the parts of the surrogate deform as they are being arrested by the vehicle or its restraint system. A simulator's kinematic behavior is determined by its body dimensions, mass distributions, and joint characteristics (that is, the body joint's articulation and resistance to motion). Its impact behavior is a function of the mass distribution plus the shape and the deformability of the body parts. These aspects of the simulator are discussed in the next four sections.

6.1.1 BODY DIMENSIONS AND WEIGHT DISTRIBUTION—To achieve fidelity in these body characteristics, it is necessary that the shapes, lengths, weights, moments of inertia, and c.g. locations of the surrogate be consistent with its human counterpart. This correspondence must apply to the surrogate as a whole as well as to its individual segments. Fidelity in these specifications (along with proper articulation) is necessary but not sufficient to ensure that all parts of the human simulator will have the proper kinematics and load their portion of the restraint system in a human-like manner.

6.1.2 ARTICULATION—Proper articulation of body segments must accompany fidelity of body dimensions and weights if the kinematics of the surrogate are to be accurate in dynamic events. Such articulation requires that body joints be appropriately simulated in their location, degrees of freedom, ranges of motion, and

resistance to motion. The human joint often has a complex construction which allows for sliding of adjacent parts in addition to pivoting. Rotation often is accommodated in more than one plane as well. It may not be necessary to completely duplicate all of these types of motion in constructing an acceptable human simulator. However, no general rules can be laid down at this time as to which characteristics are needed and which are superfluous. Each surrogate development program needs to consider each joint on an individual basis until the biomechanics community obtains more information in this area. This same consideration would apply to the resistance to motion of the joint. Data are currently being gathered for both the flaccid cadaver and the braced volunteer. It is not yet clear which condition, if either, is most appropriate for a human simulator.

6.1.3 DEFORMABILITY—Deformability is used here to refer to the response of body regions under dynamic loading. This parameter is instrumental in determining the accelerations and deflections of each segment during impact. Thus, deformability can be a primary determinant of the magnitude of the injury indicators that are measured. The deformability properties can also control the size and shape of the contact areas of the surrogate against the restraint system and so affect the overall realism of the test.

6.1.4 SHAPE—The shape of the human simulator must be a close approximation to the human to assure that it has the proper geometric interfacing with the restraint system. This concept is best illustrated by the following examples:

a. Load Sharing Between Body Segments: An overly prominent rib cage may receive too large a share of the restraint system load while, as a result, the shoulders receive too little.
b. Fidelity of Contact Areas: A knee which is overly pointed or narrow could too readily penetrate a restraining panel and generate unrealistic femur loads.
c. Geometric Interferences: An improperly shaped pelvis will not engage a lap belt properly and could invalidate assessment of submarining (where the lap belt slides up over the pelvis and loads the abdomen).

6.2 Calibration of Surrogates—Some of the inadequacies of a particular human simulator can be overcome by calibration. Unfortunately, this concept has important weaknesses and must be carefully utilized. The calibration theory holds that the instrumented human simulator should be exposed to an impact which produces a known level of injury to humans. The injury indicator levels derived from the instrumented surrogate under this condition are then assumed to correlate with the degree of human injury (even though the injury indicator level will differ from that which may have been observed for an instrumented human). Two broad classes of impact exposures have been employed for calibration purposes. These are basic laboratory biomechanics tests and some special types of field accidents.

6.2.1 USE OF BASIC BIOMECHANICAL TESTS—The basic biomechanics tests used for surrogate calibration are the ones from which the injury criteria were originally derived. The disadvantages of this scheme are the modest number of test conditions that are known to produce specific human injury levels and the limited correspondence between these test conditions and automobile crash exposures. A major function of an injury criterion is to enable estimation of the severity of impacts that differ from the data base from which the criterion was derived. However, since the current data base is limited, any number of criteria can be made consistent with it. Such hypothesized criteria may not predict injury reliably for impact wave forms that are different from the data base. These difficulties are compounded by the fact that the surrogate will typically be employed to evaluate impacts that are quite different from the basic biomechanics tests in impactor shape, loading site, and direction of loading.

6.2.2 RESTAGING OF FIELD ACCIDENTS—The second calibration alternative is the restaging of field accidents. Typically, the field collision is restaged by using vehicles, crash orientations and impact speeds that match those in the field accident. If the laboratory test duplicates the actual vehicle damage patterns and vehicle post-crash kinematics, there is a reasonable assurance that the re-created impact event matches the field crash. Of course, this assurance applies only to the vehicles and does not necessarily apply to the occupants.

6.2.2.1 *Positioning of Surrogate*—An area of concern in the restaging of accidents is the uncertainty in matching the surrogates' positioning in the vehicle to that of the human occupant. The latter's exact pre-impact position is generally unknown; yet, crash test experience has indicated that small differences in dummy placement can often produce substantial differences in the level of the measured injury indicators. Some consideration has been given to the concept of matching the vehicle interior damage caused by the surrogate to that caused by the crash victim. The drawbacks here are the number of trial-and-error laboratory tests needed to achieve the damage duplication and the unknown sensitivity of panel damage as a monitor of impact severity.

6.2.2.2 *Diversity of Accident Victims*—A second restaging dilemma is the diversity in sizes and responses of field accident victims. At the present time, only the 50th percentile adult male dummy has been developed to any extent. Few of the field accidents will have injured occupants who will correspond to this dummy size. It is also to be expected that there would be a large spread in injuries received by actual crash victims in identical accidents due solely to innate variations of tolerances among individuals. Thus, a large number of similar field accidents would be needed to establish the human injury level that would be respresentative of a given impact condition.

6.2.2.3 *Biofidelity of Surrogate*—Finally, overshadowing this approach to calibration there are the biofidelity concerns expressed previously. The surrogate must be reasonably human-like in the characteristics of dimensioning, weight distribution, articulation, deformability, and shape; otherwise, it will simply not engage restraint systems as a human would. No amount of calibration can offset a basically improper interaction between the human simulator and the vehicle environment.

6.2.3 COMPUTER RECONSTRUCTION OF FIELD ACCIDENTS—Computer models have been developed to reconstruct the kinematics of a vehicle in some of the simpler types of field accidents (Ref. 94). A model of this type is being used currently to augment field accident data files by providing estimates of the collision severity for certain types of accidents. Computer models are also available to simulate the behavior of a vehicle occupant given the vehicle's deceleration history and the characteristics of its passenger compartment (Ref. 95).

6.3 Use of Surrogates in Automotive Testing—Paragraphs 6.1 and 6.2 describe a number of problems that can arise from the use of imperfect human surrogates and test practices. However, it should be recognized that current test surrogates and methods may suffice for the majority of laboratory work which involves evolutionary development of occupant protective systems. These revised systems can often be assessed on a relative basis by comparing their performance to that of current systems[10]. If there is a need to evaluate an occupant protection system that is significantly different from established practice, then the automotive tester should emulate the biomechanics researcher and consider the employment of animal, cadaver, or volunteer testing to carry out a more complete performance evaluation. Advanced test devices are being developed worldwide and some of these may be useful for investigating a specific problem area. SAE is sponsoring five task forces [11,12] which are developing guidelines for creating better occupant simulators and injury criteria. Other research groups, under government or industry sponsorship are developing fundamental performance criteria upon which more human-like surrogates can be based. All of these efforts can reasonably be expected to lead to important advancements in the future construction and use of occupant simulators.

7. Notes

7.1 Marginal Indicia—The change bar (l) located in the left margin is for the convenience of the user in locating areas where technical revisions have been made to the previous issue of the report. An (R) symbol to the left of the document title indicates a complete revision of the report.

10. Current systems can be evaluated by field accident data if the system has been in production in sufficient volume over sufficient time.
11. Serving under the guidance of the Human Biomechanics and Simulation Subcommittee.
12. The Human Mechanical Response Task Force has published SAE J1460 MAR85 entitled "Human Mechanical Response Characteristics."

PREPARED BY THE SAE HUMAN INJURY CRITIERIA TASK FORCE OF
THE SAE MOTOR VEHICLE SAFETY SYSTEMS TESTING COMMITTEE

Clinical Response of Human Subjects to Rear-End Automobile Collisions

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ABSTRACT. Brault JR, Wheeler JB, Siegmund GP, Brault EJ. Clinical response of human subjects to rear-end automobile collisions. Arch Phys Med Rehabil 1998;79:72-80.

Objective: Forty-two persons were exposed to controlled low-speed rear-end automobile collisions to assess the relation between both gender and impact severity and the presence, severity, and duration of whiplash-associated disorders (WAD). Individual measures were also assessed for their potential to predict the onset of WAD.

Design: Experimental study subjecting individuals to a speed change of 4km/h and 8km/h and utilizing pretest and posttest physical examinations (immediately after and 24 hours after impact) to quantify subjects' clinical response.

Results: Approximately 29% and 38% of the subjects exposed to the 4km/h and 8km/h speed changes, respectively, experienced WAD symptoms, with cervical symptoms and headaches predominating. Objective clinical deficits consistent with WAD were measured in both men and women subjects at both 4km/h and 8km/h. At 4km/h, the duration of symptoms experienced by women was significantly longer when compared with that in men ($p < .05$). There were no significant differences in the presence and severity of WAD between men and women at 4km/h and 8km/h or in the duration of WAD at 8km/h. There was also no significant difference in the presence, severity, and duration of WAD between 4km/h and 8km/h. No preimpact measures were predictive of WAD.

Conclusion: The empirical findings in this study contribute to establishing a causal relationship between rear-end collisions and clinical signs and symptoms.

APPROXIMATELY 6.5 million motor vehicle accidents occurred in the United States in 1994, resulting in approximately 3.2 million injuries. Eighteen percent of the accidents involving passenger cars were rear-end impacts that caused injury to 500,000 persons.[1] Rear-end impacts result in a higher frequency of "whiplash" injuries in comparison with other crash configurations.[2-4] According to the Insurance Research Council,[5] the incidence of bodily injury liability claims associated with motor vehicle accidents has been increasing, with sprain/strain injuries accounting for the greatest share of injuries. Frequently, scientific literature uses the term *whiplash* to describe not only a mechanism of injury but also the injury or syndrome associated with the mechanism. According to the recommendations proposed by the Quebec Task Force in 1995, this study uses the term *whiplash-associated disorders* (WAD) to refer to the various clinical manifestations associated with the whiplash injury mechanism, including cervical soft-tissue strain, headache, dizziness, tinnitus, memory loss, temporomandibular joint (TMJ) pain, and others.[6]

Because of the prevalence of this condition in low-speed rear-end automobile collisions, much research has attempted to identify who is at risk for the disorder. Epidemiologic studies[2,4,6-10] have concluded that women sustain WAD more frequently than do men. States and coworkers[2] showed that women have a greater ratio of head mass (represented by head circumference^3) to neck cross-sectional area (represented by neck circumference^2) in comparison with men and proposed that this head-to-neck (H^3/N^2) ratio may explain the increased incidence of WAD. Using mathematical modeling based on anthropometric data, Snyder and colleagues[11] concluded that decreased cervical strength may be responsible for the increased incidence of cervical injuries in women exposed to rear-end collisions.

In the search for predictive factors, researchers have investigated the association between vehicle or crash-related factors and WAD injury severity following rear-end collisions.[12-14] In a descriptive case series study, Radanov and colleagues[14] found no correlation between the patient's perception of the severity of the automobile collision and the duration of pain. Likewise, Olsson and coworkers[12] found no correlation between the duration of neck symptoms and impact severity in rear-end collisions; however, the duration of neck symptoms increased if the estimated horizontal distance between the occupant's head and the vehicle's head restraint exceeded 10cm at the time of the collision. Using five measures of neck strain severity and two measures of impact severity, Ryan and coworkers[13] concluded that initial severity of neck strain is positively correlated with impact severity.

It is presumed within the medical community that WAD involve soft tissues, and that clinical examination often detects only subjective findings. Moreover, because the disorder is nonfatal and usually does not require surgical treatment, no pathologic studies have identified the site or nature of the offending lesions.[15] Current physical models (anthropometric devices and cadavers[16]) and mathematical models[17] typically employed by automotive safety laboratories can be useful to study general impact response. They have not, however, been fully validated against human subject response during low-speed rear-end collisions. Currently, these models are not suitable for human surrogates to study injury response during low-speed, rear-end collisions. For this reason, researchers have turned to human subjects to study the injury mechanism in staged low-speed, rear-end impacts.[18-21] In these studies, human subjects were exposed to rear-end impacts with speed changes ranging from 3.2 to 10.9km/h, with WAD produced at changes in velocity of 6 to 7km/h and above. Speed change is a common measure of collision severity in vehicle occupant studies and is equal to the difference between the preimpact and postimpact velocity of the vehicle. In the situation of a stationary vehicle being rear-ended, the speed change is equal to the postimpact speed of the rear-ended vehicle.

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Given the greater frequency of WAD reported in women, a specific goal of this study was to expose an equal number of male and female volunteer human subjects of a specific age group to two controlled low-speed rear-end automotive collisions (change in velocity of 4km/h and 8km/h) to assess the relation of gender and impact severity with the presence, severity, and duration of WAD. A second purpose of this study was to assess the potential for individual parameters to predict the onset of WAD in rear-end automobile collisions. The study was also designed to assess the subjects' eventual clinical outcome after the impacts. It is hoped that our research findings will contribute to establishing the causal relationship between the whiplash injury mechanism and the objective clinical response in rear-end collisions.

METHODS

Subjects

This study was part of a larger project that analyzed occupant kinematics and kinetics in low-speed rear-end automobile collisions through the use of high-speed video, accelerometry, anthropometry, and electromyography (EMG). Forty-two volunteers recruited by local newspaper and university job-line advertisements participated in the study. Men and women were equally represented. Because injury statistics suggest a high incidence in this age group,[6] and in an attempt to select a homogenous population, subjects ranging from 20 to 40 years old were targeted (men, 26.4yrs [SD ± 4.5], women, 27.1yrs [SD ± 4.8]). The mean height of the men was 175cm (SD ± 5); the mean height of the women was 164cm (SD ± 5). Mean body weight was 75kg (SD ± 10) for the men and 62kg (SD ± 9) for the women.

Subjects had no medical conditions or history of soft-tissue disease or related syndrome. Additionally, subjects with a history of neck or back injury or having treatment within the previous 3 years and those with a current insurance claim for neck or back injuries were excluded. Only subjects in the 10th to 90th percentile heights for gender[22] (men, 165 to 185cm; women, 152 to 173cm) and 10th to 90th percentile weights for their height[22] were eligible for the study. In addition, a maximum erect seated height of 95.5cm was permissible to ensure head but no neck contact with the head restraint. This latter criterion was required to allow kinetic analysis of data. Pretest magnetic resonance imaging (MRI) of the head and cervical spine, interpreted by a radiologist, ensured that no subject had a fracture, disk herniation, disk protrusion greater than 2mm, spinal canal stenosis, tumor, or any degenerative findings rated as moderate or greater. These criteria were developed to exclude subjects with MRI findings that may have predisposed them to acute injury. Subjects were asked to refrain from drastically changing their activity level in the week before testing and to not drink alcohol 24 hours before and after testing.

Test Apparatus

Subjects were seated in the right front seat of a stationary 1990 Honda Accord, which was struck on the rear bumper by a 1976 Volvo 240D station wagon. The Volvo was accelerated down a ramp from a predetermined height to impact speeds of 4.86 ± .12km/h and 10.02 ± .06km/h to produce a speed change of 3.95 ± .11km/h and 8.10 ± .11km/h, respectively, for the Honda. Both vehicles were in neutral gear with the engine off. After impact, the Honda rolled forward into gravel located about 3m ahead of the vehicle and decelerated to a stop. Speed change of the target vehicle was measured with an MEA 5th wheel[a] (commercial device for low-speed impact testing) and verified with bumper-mounted load cells.[b]

The choice of 4 and 8km/h speed changes was based on previous testing of human subjects in rear-end collisions which reported no symptoms at a speed change of 4km/h and only minimal WAD of 2 days' duration with a speed change of 8km/h.[18,19]

Human Subject Protection

Protection policies and procedures for the human subjects were in accordance with and approved by the Western Institutional Review Board. Preliminary subject eligibility was determined by an initial telephone interview, after which subjects visited the test facility where the project's principal investigator (GPS) explained the test protocol and procedures to each subject and informed consent was obtained. Subjects also completed a questionnaire assessing their availability, occupation, medical history, and physical activity. The head and neck of each subject was examined using MRI to determine eligibility based on the previously defined radiologic criteria.

Anthropometry

Anthropometric measures were obtained from each subject by a physical therapist (EJB). Head circumference was recorded via metal tape measure at the level of the glabella (most anterior protrusion of forehead) and opisthocranion (most posterior protrusion of back of head) perpendicular to the midsagittal plane. Neck circumference, also recorded via metal tape measure, was obtained at the midpoint of the neck perpendicular to the long axis. These two anthropometric measurements were used to calculate the ratio of head volume to neck cross-sectional area (computed using H^3/N^2).

Pretest Clinical Examination

At least 48 hours before the collision testing, maximum isometric cervical flexion and extension force was measured by a physical therapist with a MicroFET[c] hand-held dynamometer. Maximum isometric cervical flexion was measured with the subject supine on an examination table with the head and neck in a neutral position, C7 placed at the edge of the table so that the head and neck were extended over the edge, arms folded across the chest, and the torso stabilized. The dynamometer was centered on the glabella, and the subject was instructed to maintain maximally the head-neutral position while a downward force was applied to the forehead. Maximum isometric cervical extension was measured with the subject prone, positioned with the sternoclavicular joints at the edge of the table. The head and neck were over the edge of the table in a head-neutral position, hands behind the back, and torso stabilized. The subject was instructed to maintain maximally that head position while resistance was applied to the opisthocranion in a downward direction. A minimum 48-hour delay between isometric cervical testing and impact testing was chosen because the maximum isometric contractions of the cervical muscles potentially could have induced symptoms in these cervical spine muscles that mimic WAD, thus confounding our assessment of symptoms caused by the vehicle impacts. Measurement of maximum isometric cervical flexion and extension was performed to investigate whether these measures predispose individuals to WAD. In addition, the cervical flexion and extension maximal isometric forces were measured 24 hours after both impacts and compared with preimpact forces as an objective clinical measurement of cervical muscle function.

Before each impact, the physical therapist examined the cervical spine of each subject. The purpose of the clinical examinations was to use objective clinical measures to assess the subjects' neuromuscular system, to establish baseline values for

the measurements, to identify objective clinical changes in these measurements attributable to our intervention, and to identify any abnormal neuromuscular condition present either preimpact or postimpact that might require medical attention, which helped to ensure human subject protection. Components of a basic orthopedic examination of the cervical spine were performed, including measurement of cervical and TMJ active range of motion (ROM), assessment of the C4-T1 myotomes and dermatomes, deep tendon reflexes, and point tenderness.[23]

Cervical flexion, extension, right and left lateral flexion, right and left rotation, protraction, and retraction active ROM were measured with the cervical range of motion device (CROM),[d] a plastic instrument that rests on the bridge of the nose and ears and that is secured at the back of the head with Velcro. The CROM had three orthogonal goniometers to measure sagittal, frontal, and coronal plane motions and a bubble level and ruler for measuring protraction/retraction. Previous studies have demonstrated its intratester reliability.[24,25] Subjects were seated in a straight-back chair with the feet flat and hands on the lap, facing and focusing forward, with a lumbar support to maintain the lumbar lordosis. The CROM was applied to the head and the subjects given verbal and visual instructions on the performance of each cervical movement. Subjects were allowed one to two practice trials for each motion followed by the experimental trial, which was documented.

Range of motion of the TMJ was measured with the subject in a seated position. The examiner instructed the subject in maximum jaw opening, left excursion, and right excursion, and measurements were made in millimeters with a ruler.

Assessment of the C4-T1 dermatomes was done to evaluate sensation of cervical nerve roots emanating to the upper extremities. This was accomplished by performing a sharp/dull test with a pinwheel. With the subject seated, eyes closed, and the hands supinated on the lap, the right and left C4-T1 dermatomes were given either a sharp or dull sensation via the alternate ends of a pinwheel, and the subject was asked to answer appropriately.

The C4-T1 myotomal strength was assessed bilaterally using manual muscle testing (MMT) with the MicroFET dynamometer following published guidelines.[26] The C4 myotome, represented by the upper trapezius muscle, was tested by measuring the subjects' maximum isometric force output at 90° of arm elevation in the scapular plane. Arm elevation has been shown to recruit maximally the upper trapezius.[27] The C5/6 myotomes, represented by the elbow flexors, were tested by measuring maximum isometric elbow flexion. The C7 myotome, represented by the triceps muscle, was assessed by measuring subjects' maximum isometric elbow extension. The C8/T1 myotomes, represented by the first dorsal interosseous muscle, were assessed by measuring maximum isometric abduction of the index finger. All of the MMT with the hand-held dynamometer was performed with the subject seated on an examination table, except the test for the C7 myotome, which was performed with the subject supine on a mat to give the examiner a mechanical advantage.

Deep tendon reflexes of the bilateral brachioradialis and triceps were measured with a reflex hammer and graded on the established 1+ to 4+ scale.[28] Point tenderness of specific regions of the body was assessed by the examiner through palpation of the soft tissue overlying the occiput and suboccipital area, as well as the bilateral sternocleidomastoid (SCM), scalene, cervical paraspinal, upper trapezius, and middle trapezius muscles and TMJ. Subjects were seated in a chair and instructed by the examiner to relax as the specific regions were palpated. The examiner asked the subjects to report any tenderness elicited by the palpation, which was graded on a 1+ to 4+ scale (1+ = slight, 2+ = minimal, 3+ = moderate, 4+ = severe).

Electromyography

Surface EMG electrodes were applied to the subject's bilateral SCM and cervical paraspinal muscles by an investigator experienced in the use of surface EMG (JRB). The primary purpose of the EMG sampling was to investigate cervical muscle response during the impact (EMG results will be presented in another report). A secondary purpose of the EMG was to ensure that preimpact cervical muscle response, which could alter the occupant kinematics during impact, did not increase above seated resting levels. Four-millimeter IVM Ag-AgCl reusable disk electrodes[e] were taped to the skin overlying the clavicular head of the bilateral SCM muscle in a bipolar configuration using the technique of Zipp.[29] Eight-millimeter IVM Ag-AgCl reusable disk electrodes[e] were taped to the skin overlying the posterior cervical spine in a bipolar configuration 1cm lateral to the C4 and C6 spinous processes bilaterally with a 4cm interelectrode distance. To reduce impedance at the electrode sites, the skin was shaved, lightly abraded with sandpaper, wiped with 50/50 alcohol/distilled water, and coated with electrode gel.[30] The electrode leads were connected to a transmitter.[f] The EMG signals were telemetered to a receiver, converted from analog to digital by an A/D board,[g] and then analyzed by LABVIEW[g] software. Proper placement of the electrodes was confirmed by visual inspection of the muscle interference pattern during manual muscle testing. EMG data were sampled at a frequency of 1,000Hz and band-pass–filtered with a second-order Butterworth filter with high- and low-pass filters of 40 to 500Hz, respectively.

Collision 1

Following the initial examination, subjects were exposed to their first test collision. To control for series effect, the order of impact severity was randomized by flipping a coin. Subjects were not told which speed change they would experience.

In the test vehicle (Honda), the fore/aft seat position, seat back angle, and head restraint height were kept constant for all subjects. Subjects were instructed to sit with their backs against the seat back, face forward with the head horizontal, feet on the floor, hands in the lap, and to otherwise assume a normal seated position. The lap/shoulder belt was worn by all subjects, and the subjects were instructed not to rest their heads on the head restraint. The horizontal distance between the opisthocranion and head restraint was measured with a FaroArm 3D digitizer[h] and confirmed from the digitized video frame corresponding to impact.

Ryan and coworkers[31] found that automobile occupants who were unaware of the impending collision were 15 times more likely to have persisting WAD 6 months postimpact; therefore, we attempted to control for this variable by minimizing subject expectation of the impending collision. First, subjects did not see the striking vehicle (Volvo) on the ramp prior to their test collisions and therefore derived no preconception of impact severity from its height on the ramp. Second, a curtain was placed between the Honda and Volvo to eliminate visual cues of the impending collision, and no test personnel were visible to the subjects in the minutes immediately preceding the test collision. Third, subjects wore foam ear plugs, and loud music was played before and during the test collisions to eliminate aural cues. Fourth, the subjects' seated position was monitored before impact via closed-circuit video from a camera mounted to the Honda's driver's side A-pillar to ensure no change of position prior to impact. Lastly, the subjects' EMG output from the bilateral SCM and cervical paraspinal muscles was visually monitored for a minimum of 1 minute before impact to ensure

no increase in muscular contraction above that present in the normal seated position.

After the first test collision, subjects were told the severity of the rear-end collision to minimize apprehension in those subjects first exposed to the 8km/h speed change and who might elect to withdraw if they presumed the first collision was a 4km/h speed change.

Posttest Clinical Examination

A second clinical examination was performed within 30 minutes after the first test collision. All of the tests performed in the preimpact clinical examination were repeated by the same examiner, and subjects were asked whether they were experiencing any symptoms not present before the impact. Because the primary symptom of WAD is often the subjective expression of pain,[6] the objective of the examination was to quantify the subjects' symptoms to treat the data statistically. Therefore, if subjects reported symptoms following a test collision, the postimpact examination included the McGill Pain Questionnaire, which was designed to provide quantitative measures of clinical pain that can be treated statistically.[32] Through the use of word descriptors chosen by subjects to best represent their pain, the quality of pain was documented. The intensity of the pain was assessed with the questionnaire's Present Pain Intensity scale (PPI), which is a combination numerical and descriptive measure of pain. The PPI was recorded as a number from 1 to 5, with each number associated with the following words: 1 = mild, 2 = discomforting, 3 = distressing, 4 = horrible, and 5 = excruciating. Subjects recorded the location of pain on the questionnaire's body diagram, which portrayed an anterior and posterior outline of a body of appropriate gender. Subjects were instructed to shade the area on the body diagram corresponding to the location of their pain. Because the subjects could not be observed continuously after the test collisions, the assessment of the subjects' pain experience included the McGill Home Recording Card.[32] After each test collision, the subjects were given a card on which they recorded their symptoms and level of pain (PPI) at four different times throughout the day (morning, noon, dinner, bedtime) between the time of their first postimpact examination and the 24-hour follow-up examination. The Home Card provided a method of documentation away from the test site and allowed continuous tracking of symptoms and injury severity.

24-Hour Posttest Examination

Subjects returned to the test facility the day after the test collision (approximately 24 hours posttest) for a third clinical examination during which all of the previous cervical spine testing was repeated. If symptoms were reported, the McGill Pain Questionnaire was completed. In addition, subjects returned the completed McGill Home Recording Card and were observed until their symptoms and clinical deficits subsided.

Collision 2

Subjects returned for the second test collision a minimum of 7 days after the first test collision or 7 days after resolution of their symptoms attributed to the first test collision. The protocol for the second test collision was identical to that in the first, except that subjects were aware of the severity of the second test collision beforehand.

Statistical Analysis

The null hypothesis was that there would be no difference in the presence, severity, and duration of WAD between genders and impact severity. Comparisons of the presence, severity, and duration of WAD between genders were performed using Fisher's exact test,[33] Wilcoxon rank sums test,[34] and log-rank test,[35] respectively. The tests were performed on the 4km/h and 8km/h data separately. Comparison of the presence of WAD between impact speeds was carried out using McNemar's chi-squared test.[36] Comparisons of the severity and duration of WAD between speed changes were carried out using the Wilcoxon sign-rank test.[34] Comparisons of severity and duration were performed using only the data of subjects with WAD.

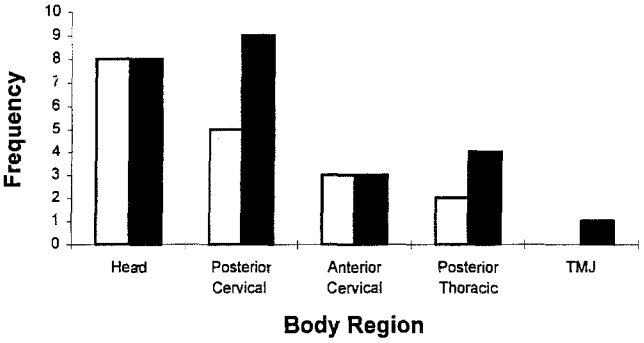

Fig 1. Symptom distribution for the men and women subjects combined: □, 4km/h; ■, 8km/h.

Logistic regression[37] (modeled using a log linear model) was used to determine whether the measured variables of H^3/N^2, pre-impact cervical flexion and extension isometric force, pre-impact cervical ROM, and horizontal distance from head to head restraint could be used to predict the onset of WAD. The likelihood ratio chi-squared was used to test for significant effects of the various measurements. A separate regression analysis was carried out for the 4km/h and 8km/h trials.

A repeated measures analysis of variance (ANOVA)[38] was used to test the effect of the presence of symptoms and the time of measurement (preimpact, postimpact, 24-hours postimpact) on ROM and cervical MMT values. This analysis was conducted on the cervical ROM values of flexion and extension, right and left lateral flexion, right and left rotation, and protraction and retraction to test for statistical significance. Again, the analyses were performed separately for the 4km/h and 8km/h trials. A p value of .05 level of significance was used for all statistical comparisons.

Power was calculated for the duration of symptoms between the 4km/h and 8km/h collisions, assuming the observed values in this study are exponentially distributed. Under this assumption, the power of the study to detect a significant difference in duration of symptoms is 90%.

RESULTS

Because no women at the 4km/h and only one woman at the 8km/h impact severity experienced symptoms at the time of either of the postimpact clinical examinations, the data from the component of the McGill Pain Questionnaire using word descriptors of pain were not utilized for statistical purposes. Given that nearly all of the symptoms experienced by the subjects occurred while the subjects were away from the test site, data obtained from the PPI index on the McGill Home Card and Pain Questionnaire were treated statistically.

Figure 1 shows the combined male and female distribution of WAD by body region for the 4km/h and 8km/h impacts. Headaches and posterior neck symptoms were the predominating complaints. After the 4km/h impact, the examiner identified three subjects with 1+ to 3+ tenderness with a duration of up to 48 hours. The 8km/h impact produced eight subjects with

Table 1: Comparison Between Genders at 4km/h

	Men	Women	*p* Value
Presence*	5 (23.8)	7 (33.3)	.73†
Severity‡	1.2 (1.1)	1.3 (1.4)	1.00§
Duration‖	2 (1-8)	12 (1-48)	.03¶

* Count (percent).
† Fisher's exact test.
‡ Mean (SD) PPI of subjects with symptoms.
§ Wilcoxon rank sums test.
‖ Median duration (range) in hours of subjects with symptoms.
¶ Log-rank test.

1+ to 2+ tenderness lasting up to 48 hours. In both impacts, the areas of tenderness included the bilateral cervical paraspinal, SCM, scalene, upper trapezius, middle trapezius, and occiput areas. No significant differences were noted postimpact for the reflex, sensory, or upper extremity MMT.

At 4km/h, there were no significant differences in either the presence of WAD between men and women or in the mean severity (PPI) of symptoms between men and women (table 1). A significant difference, however, did exist in the median duration of symptoms between men (2 hours) and women (12 hours). An unpaired *t* test between male and female H^3/N^2 ratio revealed a statistically significant higher ratio in women in our sample population. At 8km/h, no significant differences existed in the presence, severity, or duration of WAD between men and women (table 2). There were no differences in the presence, severity, or duration of WAD between the 4km/h and 8km/h collisions when both men and women were combined (table 3).

The logistic regression analyses failed to identify a pre-impact measurement variable predictive of the presence of WAD at either 4 or 8km/h (tables 4 and 5, respectively).

Analysis of the effect of the 4km/h impact severity on ROM measurements over time revealed that, at both postimpact examinations, subjects with and without symptoms had a significant decrease in cervical flexion, extension, retraction, and right lateral flexion, with left lateral flexion ROM approaching statistical significance (table 6). In addition, an analysis of subjects with and without symptoms revealed that the subjects with symptoms had significantly reduced left and right lateral flexion ROM values preimpact and at both postimpact examinations (table 6). At the 8km/h impact severity, ROM values were significantly reduced immediately postimpact for cervical flexion, right and left lateral flexion, right rotation, and retraction for both groups with and without symptoms (table 7). An analysis of the effect of the interaction between the presence of symptoms and the time of examination on ROM values at both the 4km/h and 8km/h impact severity revealed no significant differences (tables 6 and 7).

Significantly higher cervical flexion MMT values were measured postimpact in subjects with and without symptoms at both 4 and 8km/h, but there was no difference in cervical MMT values when the groups were analyzed by the presence of symptoms.

Three subjects (one male and two female) having experienced only the 4km/h trial were lost to observation for the 8km/h trial. No increase in cervical EMG activity was observed immediately preimpact in comparison with the resting levels, suggesting not only adequate control of the potential confounding variable of a preimpact increase in cervical muscle resistance but also that the subjects were unprepared for the collisions.

Table 2: Comparison Between Genders at 8km/h

	Men	Women	*p* Value
Presence*	8 (38.1)	7 (36.8)	1.00†
Severity‡	0.8 (0.6)	0.9 (0.4)	1.00§
Duration‖	8.8 (0.3-111)	24 (0.5-24)	0.91¶

* Count (percent).
† Fisher's exact test.
‡ Mean (SD) PPI of subjects with symptoms.
§ Wilcoxon rank sums test.
‖ Median duration (range) in hours of subjects with symptoms.
¶ Log-rank test.

Table 3: Comparison of 4km/h With 8km/h

	4km/h	8km/h	*p* Value
Presence*	12 (28.6)	15 (38.5)	.17†
Severity‡	1.3 (1.2)	0.8 (0.5)	.99§
Duration‖	5.8 (1-48)	24 (0.3-111)	.14§

* Count (percent).
† McNemar χ^2 test.
‡ Mean (SD) PPI of subjects with symptoms.
§ Wilcoxon sign-rank test.
‖ Median duration (range) in hours of subjects with symptoms.

DISCUSSION

The results of this study reveal objective clinical deficits consistent with WAD at both 4km/h and 8km/h speed changes in both men and women. The distribution of symptoms by body region agrees with clinical reviews and descriptive case series of WAD,[6,14,15] with cervical symptoms and headaches predominating. A potential confounding variable which may have influenced the number of complaints of headache was the tightness of the head gear worn to measure head acceleration. The WAD experienced by the subjects is consistent with the Quebec Task Force clinical classification of grade II, which is defined as neck complaints and musculoskeletal signs such as decreased ROM and point tenderness.[6]

The lack of a significant difference in the presence of WAD between male and female subjects is inconsistent with many previous reports documenting an increased incidence of WAD in females.[2,4,6-10] These previous epidemiologic studies, utilizing clinical reviews of patients, emergency room medical records, insurance claims, and traffic collision reports, have incorporated a wide range of impact severity. Perhaps the speed changes in our study were too low, narrow, or both to delineate a difference between genders. In a recent questionnaire-based study of 202 victims of rear-end collisions,[39] no significant difference was evident in the prevalence of chronic neck pain or headache between males and females. That study, however, investigated

Table 4: Logistic Regression of Factors Used to Predict for the Presence of WAD Following a 4km/h Collision

	Subject Without Symptoms	Subject With Symptoms	
	Mean (SD)	Mean (SD)	*p* Value*
Head to head rest (mm)	44.0 (20.1)	49.1 (15.4)	.44
H^3/N^2 (cm)	152.7 (22.70)	156.5 (24.27)	.63
Cervical MMT (Newtons)†			
Flexion	104.2 (46.6)	81.6 (43.2)	.14
Extension	217.9 (62.8)	193.4 (67.3)	.26
Cervical ROM (°)‡			
Flexion	61.0 (6.7)	58.0 (8.4)	.23
Extension	69.9 (7.6)	70.5 (8.8)	.81
R lateral flexion	45.3 (6.6)	40.7 (7.4)	.06
L lateral flexion	46.5 (6.2)	43.3 (5.3)	.13
R rotation	68.9 (7.6)	67.7 (8.4)	.63
L rotation	68.7 (6.7)	66.0 (4.7)	.21
Retraction	3.3 (1.1)	3.1 (0.9)	.68
Protraction	3.7 (0.9)	3.8 (1.4)	.78

* Likelihood ratio χ^2 from logistic regression.
† Pretest cervical manual muscle test.
‡ Pretest cervical range of motion.

Table 5: Logistic Regression of Factors Used to Predict for the Presence of WAD Following a 8km/h Collision

	Subject Without Symptoms	Subject With Symptoms	
	Mean (SD)	Mean (SD)	*p* Value*
Distance to head rest (mm)	41.7 (18.3)	40.9 (18.5)	.90
H^3/N^2 (cm)	153.3 (21.4)	154.7 (24.9)	.84
Cervical MMT (Newtons)†			
Flexion	100.3 (47.2)	97.6 (44.1)	.88
Extension	217.5 (65.1)	211.2 (56.6)	.98
Cervical ROM (°)‡			
Flexion	61.5 (7.4)	59.3 (6.4)	.35
Extension	69.8 (8.6)	71.9 (8.2)	.44
R lateral flexion	45.2 (7.4)	41.7 (5.7)	.14
L lateral flexion	46.6 (7.3)	44.5 (6.7)	.38
R rotation	70.8 (8.0)	67.7 (7.3)	.23
L rotation	68.1 (6.4)	66.1 (5.9)	.34
Retraction	3.0 (1.0)	3.2 (0.9)	.24
Protraction	3.8 (1.3)	4.0 (1.2)	.70

* Likelihood ratio χ^2 from logistic regression.
† Pretest cervical manual muscle test.
‡ Pretest cervical range of motion.

chronic symptoms and not initial symptoms, precluding any direct comparison to our results.

The presence of WAD at the 4km/h speed change conflicts with all previously published accounts of low-speed rear-end automobile testing involving human subjects.[18-21] The different results found in our study may be explained by the small sample size, predominately male population, variable age range, variable speed changes,[18,19,21] multiple impact exposures, and failure to perform a complete pre-impact and postimpact objective clinical examination in the previous studies. In addition, in all cases, the subjects were either the investigators or professionally associated with the investigators, which could potentially introduce researcher bias. Although these studies provide the groundwork for understanding occupant response to low-speed rear-end impacts, the results are difficult to apply to any subset of the general population given these limitations.

Although the median duration of symptoms at 4km/h was significantly longer for women in comparison with men (12 hours versus 2 hours), no clinical significance can be deduced from this information given the fact that few persons would be expected to seek medical treatment beyond an initial consultation for symptoms of such short duration.

The failure to demonstrate a difference between the 4km/h and 8km/h trials with respect to the presence, intensity, and duration of WAD is noteworthy, because one might expect greater injury as speed change increases. Perhaps the range of speed change was too low and narrow to elicit such a biologic effect in our subjects. Increasing the range of impacts is not a viable option, however, given the potential harm associated with exposing human subjects to greater impact severity. Previous studies have failed to reveal a dose-response for duration[12] and severity[14] of WAD following rear-end collisions. However, the studies included higher speed collisions than used in our study, and the measure of accident severity used by Radanov and coworkers,[14] the patient's perception, could be considered unreliable.

The inability of the H^3/N^2, cervical flexion and extension isometric force, and horizontal distance from head to head restraint to predict the presence of WAD failed to support several theories[2,11,12] which purport that preimpact parameters may influence an individual's susceptibility to WAD. Differing study

Table 6: ANOVA for Range of Motion Values at 4km/h

	Time Period	No Symptoms*	Symptoms*	Symptom Effect†	Time Effect†		Symptom × Time Effect†
Flexion (°)	Pretest	61.0 (6.7)	58.0 (8.4)	.21	.004‡	Posttest < pretest	.51
	Posttest	59.9 (7.0)	56.2 (9.0)			Posttest < post-24hrs	
	Post-24hrs	61.3 (6.6)	59.0 (8.8)				
Extension (°)	Pretest	69.9 (7.6)	70.5 (8.8)	.90	.02	Posttest < pretest	.88
	Posttest	68.6 (7.8)	68.7 (7.3)			Posttest < post-24hrs	
	Post-24hrs	70.0 (7.3)	70.3 (10.3)				
R lateral flexion (°)	Pretest	45.3 (6.6)	40.7 (7.6)	.04‡	.03‡	Posttest < pretest	.57
	Posttest	44.4 (5.4)	40.0 (6.8)			Posttest < post-24hrs	
	Post-24hrs	45.9 (6.6)	40.7 (8.5)				
L lateral flexion (°)	Pretest	46.5 (6.2)	43.3 (5.3)	.04‡	.06		.28
	Posttest	46.5 (5.7)	41.7 (6.8)				
	Post-24hrs	47.7 (6.3)	43.2 (7.6)				
R rotation (°)	Pretest	68.9 (7.6)	67.7 (8.4)	.38	.56		.16
	Posttest	68.9 (7.0)	67.3 (6.6)				
	Post-24hrs	70.5 (7.1)	67.0 (6.6)				
L rotation (°)	Pretest	68.7 (6.7)	66.0 (4.7)	.14	.16		.67
	Posttest	68.1 (6.6)	64.7 (3.5)				
	Post-24hrs	68.4 (6.6)	65.7 (4.3)				
Protraction (cm)	Pretest	3.7 (0.9)	3.8 (1.4)	.98	.28		.67
	Posttest	3.8 (0.9)	3.9 (1.4)				
	Post-24hrs	4.0 (0.9)	3.8 (1.6)				
Retraction (cm)	Pretest	3.3 (1.1)	3.1 (1.0)	.78	.0002‡	Posttest < pretest	.19
	Posttest	3.0 (1.0)	2.8 (0.8)			Posttest < post-24hrs	
	Post-24hrs	3.4 (0.8)	3.6 (0.8)				
TMJ opening (mm)	Pretest	47.5 (7.2)	45.3 (4.3)	.34	.17		.06
	Posttest	47.5 (7.5)	45.7 (4.5)				
	Post-24hrs	47.6 (7.2)	45.2 (3.9)				
TMJ L excursion (mm)	Pretest	3.5 (1.4)	4.4 (1.6)	.03‡	.28		.46
	Posttest	3.4 (1.3)	4.5 (1.6)				
	Post-24hrs	3.3 (1.3)	4.4 (1.6)				
TMJ R excursion (mm)	Pretest	3.6 (1.7)	4.1 (1.0)	.41	.53		.73
	Posttest	3.6 (1.8)	4.0 (1.0)				
	Post-24hrs	3.6 (1.7)	4.0 (1.0)				

* Mean (SD) ROM.
† *p* value.
‡ $p < .05$.

Table 7: ANOVA for Range of Motion Values at 8km/h

	Time Period	No Symptoms*	Symptoms*	Symptom Effect†	Time Effect†		Symptom × Time Effect†
Flexion (°)	Pretest	61.5 (7.4)	59.3 (6.4)	.23	.04‡	Posttest < pretest	.62
	Posttest	60.7 (7.4)	57.3 (6.0)			Posttest < post-24hrs	
	Post-24hrs	61.8 (7.3)	59.5 (6.2)				
Extension (°)	Pretest	69.6 (8.6)	71.9 (8.2)	.47	.03		.92
	Posttest	69.1 (9.0)	71.1 (7.3)				
	Post-24hrs	69.9 (7.8)	71.6 (8.7)				
R lateral flexion (°)	Pretest	45.2 (7.4)	41.7 (5.7)	.07	.01‡	Posttest < post-24hrs	.36
	Posttest	45.2 (5.8)	40.4 (6.2)				
	Post-24hrs	46.2 (6.3)	42.9 (7.4)				
L lateral flexion (°)	Pretest	46.6 (7.3)	44.5 (6.7)	.34	.0001‡	Pretest < post-24hrs	.39
	Posttest	45.6 (6.0)	42.8 (6.2)			Posttest < pretest	
	Post-24hrs	48.0 (5.7)	46.9 (7.5)			Posttest < post-24hrs	
R rotation (°)	Pretest	70.8 (8.1)	67.7 (7.3)	.23	.04‡	Posttest < post-24hrs	.68
	Posttest	69.6 (7.7)	67.1 (6.6)				
	Post-24hrs	71.4 (8.6)	68.0 (7.1)				
L rotation (°)	Pretest	68.1 (6.4)	66.1 (5.9)	.24	.65		.29
	Posttest	68.4 (6.1)	65.3 (5.1)				
	Post-24hrs	68.1 (5.9)	66.7 (4.5)				
Protraction (cm)	Pretest	3.8 (1.3)	4.0 (1.2)	.38	.6		.57
	Posttest	3.8 (1.2)	4.2 (1.1)				
	Post-24hrs	3.8 (0.9)	4.0 (1.0)				
Retraction (cm)	Pretest	2.9 (0.9)	3.2 (0.8)	.32	.0001‡	Pretest < post-24hrs	.71
	Posttest	2.9 (0.8)	3.0 (0.9)			Posttest < post-24hrs	
	Post-24hrs	3.4 (0.8)	3.7 (1.2)				
TMJ opening (mm)	Pretest	47.3 (5.4)	46.9 (7.8)	.85	.95		.48
	Posttest	47.2 (5.5)	46.9 (7.6)				
	Post-24hrs	47.4 (5.6)	46.8 (7.8)				
TMJ L excursion (mm)	Pretest	3.6 (1.4)	4.3 (1.8)	.16	.76		.49
	Posttest	3.6 (1.3)	4.2 (1.8)				
	Post-24hrs	3.5 (1.1)	4.3 (1.8)				
TMJ R excursion (mm)	Pretest	3.6 (1.9)	3.9 (1.7)	.69	.66		.07
	Posttest	3.7 (1.8)	3.7 (1.7)				
	Post-24hrs	3.6 (1.9)	3.9 (1.8)				

* Mean (SD) ROM.
† p value.
‡ $p < .05$.

designs may account for this discrepancy. In an epidemiologic survey of police accident reports, States and colleagues[2] hypothesized that a higher female H^3/N^2 ratio suggested a larger head relative to neck size and thus an increased susceptibility to WAD in rear-end automobile collisions; however, the investigators relied on military anthropometric data and did not directly measure the H^3/N^2 ratio of the subjects in their population. Our results could not be compared with those of Olsson and coworkers,[12] who reported an increased duration of neck symptoms in rear-end collisions when the head to head restraint distance exceeded 10cm, because our data did not include any head to head restraint distances greater than 8cm. Regarding the postulate by Snyder and coworkers[11] that decreased cervical strength is responsible for the greater incidence of WAD in women in rear-end collisions, no comparison can be made with our study as Snyder's group examined only neck characteristics (anthropometry, ROM, strength, and muscle reflex times) and did not expose the subjects to rear-end automobile collisions.

In our attempt to assess the outcome of clinical measurements in the subjects over time, we discovered an increase in cervical flexion isometric force postimpact, which we attributed to a learning effect resulting from the unusual nature of the activity.

Of most importance clinically was the statistically significant decrease in cervical ROM immediately following both 4km/h and 8km/h trials, which suggests that the rear-end collisions caused measurable clinical deficits. Surprisingly, these deficits occurred in subjects with and without symptoms. The presence of cervical ROM deficits after a rear-end automobile collision without concomitant symptoms may be a function of an individual's tolerance for, or perception of, pain. Although statistically significant, the magnitude of these ROM deficits was sufficiently small to be of limited clinical value. The reduced ROM measured immediately following the impacts fell within a normal range of values,[41] and, without specific knowledge of a patient's preimpact ROM values, a health care professional would not likely be capable of detecting these deficits. The CROM device was capable of measuring sagittal plane rotation, lateral flexion, and coronal plane rotation to the nearest 2°, whereas retraction and protraction could be measured to the nearest 0.5cm. Based on the standardized protocol for measurement performed by a single examiner for all subjects and the small standard deviations in ROM data, we are confident that the observed ROM deficits are real and not an artifact of the measuring device or examiner reading error.

Most interesting from a clinical perspective was the fact that the cervical ROM deficits were characteristically similar to the subjective reports of symptoms in that both were transient. In most cases, the ROM deficits were present immediately postimpact, but by the 24-hour postimpact physical examination, cervical ROM had returned to normal, consistent with most subjects' short duration of symptoms. These results are consistent with a study by Ryan and coworkers,[31] who found a strong correlation between objective measures of neck strain and the subject's own rating of neck symptoms.

The degree to which theories about causation of a disease are supported by empirical evidence is termed *construct validity*.[40] The clinical findings of this experimental study contribute to the construct validity of the whiplash injury mechanism and establish the causal relationship between the rear-end collisions and the clinical signs and symptoms. The highly variable clinical findings observed among the subjects in our study also attests to the nonspecific effect of the whiplash injury mechanism (low descriptive validity) previously reported in the literature.[40]

Our sample was one of convenience and consisted of volunteers who were selected to control for variables such as age, previous medical history, and pre-existing spine pathology. Therefore, our study population does not entirely reflect the total population exposed to rear-end automobile collisions. Furthermore, the controlled conditions under which these automobile collisions were conducted (normal seated posture, aligned impacts, head restraint available) are not representative of all rear-end collisions in a noninvestigative setting producing WAD. Additionally, we did nothing to control for other variables such as subjects' psychologic profile[42] or culture,[43] which previously have been reported to influence the outcome following the whiplash injury mechanism. Therefore, meaningful prognoses of WAD in all rear-end collisions cannot be derived from the data, because the accuracy of a given outcome is dependent on many variables.

CONCLUSIONS

Approximately 29% and 38% of individuals exposed to rear-end impacts at 4km/h and 8km/h speed changes, respectively, experienced WAD symptoms of minimal severity and short duration, with cervical symptoms and headaches predominating. The presence, severity, and duration of WAD were similar in men and women exposed to rear-end automobile collisions with speed changes of 4km/h and 8km/h. In addition, the study failed to identify useful predictive factors predisposing individuals to the development of WAD from rear-end automobile collisions. The empirical evidence gained from this experimental study contributes to the construct validity by establishing a causal relationship between rear-end collisions and clinical signs and symptoms.

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Chronic Whiplash and Whiplash-Associated Disorders: An Evidence-Based Approach

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Abstract

Whiplash is neck pain experienced as a result of a motor vehicle collision or similar trauma. Following a motor vehicle collision, 15% to 40% of patients with acute neck pain develop chronic neck pain. The cervical facet joint is the most common source of chronic neck pain after whiplash injury, followed by disk pain. Some patients experience pain from both structures. Initial management recommendations need not be directed toward an exact structural cause, but treatment includes advising the patient to remain active, prescribing medications when necessary, and providing advice regarding the generally favorable outcome. When neck pain persists, the physician should recommend medial branch blocks of the dorsal rami of the spinal nerves that supply the putative painful facet joint or joints; this is done to determine whether the facet joints are the cause of pain. When significant relief occurs on two occasions, radiofrequency neurotomy typically provides substantial relief for approximately 8 to 12 months and can be repeated indefinitely as needed. Occasionally, long-term treatment with medication may be indicated. Anterior cervical diskectomy and fusion is necessary on rare occasions.

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The term whiplash has been used to describe both a mechanism of injury (usually a motor vehicle collision [MVC]) and the neck pain caused by that injury. Whiplash-associated disorder is defined as the variety of clinical symptoms other than head, neck, and arm pain that occur after a whiplash type of trauma. In a typical rear-end MVC, the injury is caused by the abnormal biomechanics of neck motion resulting from the forward and upward motion of the torso while the head lags behind as the result of inertia. Whiplash injury is any structural damage sustained because of the whiplash forces.

Most persons involved in an MVC are not injured. Clinical studies have shown, however, that 15% to 40% of patients with acute neck pain after MVC develop chronic pain; 5% to 7% of patients become permanently partially or totally disabled.[1-5] Whiplash injury remains controversial and misunderstood, despite advances in knowledge of the biomechanical and medical aspects of this condition.

Mechanism and Biomechanics of Whiplash Injury

The whiplash event lasts well under 500 ms.[6] In a rear-end MVC, energy is transferred on impact from the bullet vehicle to the struck vehicle; the struck vehicle is then suddenly accelerated by the impact. Energy is transferred to the frame of the struck vehicle, then to the seat, and then to the occupants. The resultant biomechanics have been described.[6-9] The predominant response to the impact is forward displacement and associated upward displacement of the torso. Head motion lags behind the body as a result of the inertia of the head. The forward acceleration of the torso deforms the cervical spine into a nonphysiologic S-shaped curve, with extension developing between the lower segments and flexion developing between the uppermost segments. Most of the whiplash injury occurs during this deformation phase. The base of the skull is accelerated forward by the neck, causing the head to rotate backward and the upper cervical spine to move into extension. Overall neck extension usually stays within the physiologic range, particularly when the head is well positioned against a head restraint. As a result of head restraint contact, the head and neck rebound forward into cervical flexion.

The forces and resultant motion that cause the whiplash injury are related to neither the total range of motion of the neck nor the amount of extension or flexion—the so-called whip. Instead, it is the abnormal motion that occurs within and between individual motion segments that has the potential to injure facets, disks, and ligaments. The facet joints undergo a nonphysiologic pinching motion, with compression posteriorly and distraction anteriorly, usually coupled with shear.[10] The anulus fibrosus of the disk and longitudinal ligaments can be disrupted by the same abnormal motion.[7,8]

Facet injuries include capsular strain and tears; bony impingement; synovial fold pinching; and direct-impact injury resulting in contusion, intra-articular hemorrhage, and damage to subchondral bone.[10] Disk injuries include strain or avulsion of the anterior disk anulus, tearing of the posterior anulus, and disk herniation. Any of these structural injuries has the potential to cause acute and chronic neck pain.

The amount of force transmitted from the bullet vehicle to the struck vehicle is often reported as change in velocity. In general, the greater the change in velocity, the greater the injury to the occupants. However, this relationship is far too variable to be of use in predicting severity of injury. Moreover, data indicate that whiplash injury is better correlated with the acceleration of the struck vehicle than with the change in velocity.[11-13]

Severity of property damage is not a reliable predictor of injury or outcome in low-speed collisions. Crash characteristics and human factors are much more relevant. Crash factors include the respective size, weight, and speeds of the vehicles; the type and position of the seat and head restraint; and the ability of the vehicles to absorb or transmit energy. Human factors include the size, weight, and sex of the occupant; awareness of the impending collision; direction in which the occupant is facing at impact; and individual tissue tolerance.

Natural History of Neck Pain After Whiplash Injury

Multiple studies document the natural history of whiplash injury.[1,3,14-16] Most persons do not develop neck pain after an MVC. However, those who experience acute neck pain immediately following the MVC are three times more likely to report chronic neck pain 7 years later than are patients involved in MVCs who had no acute neck pain and patients not involved in a prior MVC.[5] In approximately 80% of patients, pain following an MVC begins the day of the collision. Of those who develop chronic pain, 20% experience delay between the time of the MVC and the onset of pain.[1,17] The literature reflects a wide range of patients who have acute neck pain after whiplash injury and who recover completely, but the consensus is between 60% and 85%.[1,3,5,14,15] Up to 40% of patients develop chronic (usually mild) pain, but 5% to 12% experience chronic moderate to severe pain; approximately 5% to 10% have permanent partial or total disability.[3-5,18]

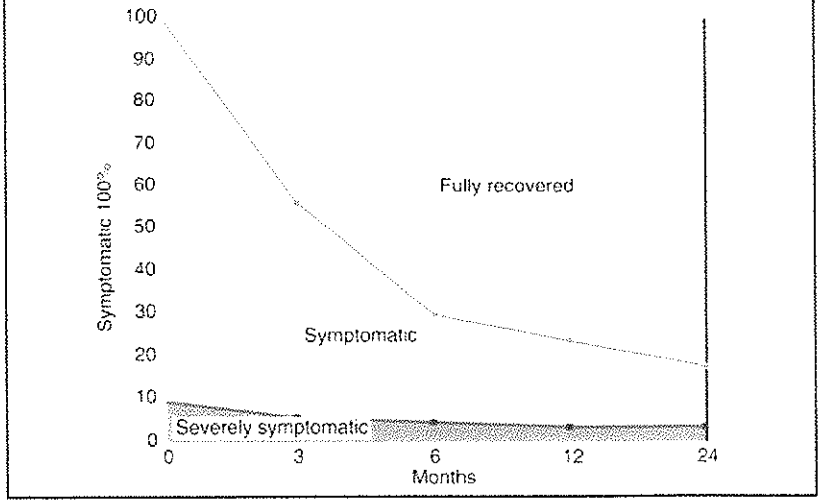
Radanov et al[3] prospectively studied 117 patients with acute whiplash. Full recovery was achieved in 56% of patients at 3 months, in 70% at 6 months, and in 76% at 12 months (Figure 1). At 24 months, 21 patients (18%) remained symptomatic; of these patients, 3 were partially disabled and 2 were totally disabled.

Prognostic Factors

General Factors

Many prospective studies have identified prognostic factors for adverse outcomes. The strongest predictor of poor outcome is high initial intensity of pain.[14,15,18] In a systematic review, Scholten-Peeters et al[15] noted that older age, female sex, high initial psychological response, and compensation or litigation were not associated with an adverse prognosis. There was limited prognostic value for the patient with pain in multiple areas or with prior psychosocial problems. Evidence was inconclusive regarding the prognostic value of head position at impact, radicular symptoms, cognitive impairment, poor concentration, prior headache, being unprepared for collision, and a change of velocity >10 km/h (>6.2 mph).[15] However, Côté et al[14] found that older age, female sex, radicular symptoms, multiple areas of pain, and being unprepared for impact portend a worse prognosis.

Figure 1



Recovery curve for acute whiplash, based on data from Radanov et al.[3] Full recovery was seen in 56% of patients at 3 months, in 70% at 6 months, in 76% at 12 months, and in 82% at 24 months.

Preinjury Psychological Factors

There is no preinjury personality that renders individuals more likely to develop chronic pain after whiplash, nor are there psychosocial factors that are useful predictors of chronicity.[19] Gargan et al[20] recorded symptoms and psychological test scores in 50 patients with acute whiplash within 1 week of MVC and then again at 3 months and 2 years. Psychological testing was normal initially in 82% of patients. At 3 months, testing was abnormal in 81%, and testing remained abnormal at 2 years in 69%. There was no correlation between the initial psychological testing and chronicity. The authors concluded that, in most instances, psychological problems were a consequence rather than a cause of pain.

Coping style may be quite important. Patients with better coping abilities have more favorable functional outcomes than do those who cope poorly.[21] The development of fear avoidance and of fear-avoidant behavior may contribute to the disability of the chronic pain.[22]

Work-Related Factors

Gozzard et al[4] examined factors that might affect employment and disability after whiplash. Forty of 586 patients (7%) had not returned to work. The strongest predictor of prolonged disability was intensity of symptoms. There was no difference in the prevalence of preinjury psychological illness in those who did or did not return to work. Hendriks et al[18] prospectively followed 125 patients with acute whiplash. At 1 year after injury, 12% were partially or totally impaired or disabled. There was no predictive value for baseline initial psychometric testing, litigation status, age, sex, differences in speed between vehicles, or type of early treatment.

Effect of Litigation on Outcome

The effect of litigation on outcome is often misunderstood. Multiple studies have demonstrated that personal injury litigation does not adversely affect the outcome of whiplash injury.[15,23-25] One possible reason for confusion might be the failure to discriminate between workers' compensation and personal injury litigation.[23] It is well-established that, as a group, injured workers tend to do worse than other patients with similar injuries.[23]

Swartzman et al[25] compared the outcomes of whiplash patients in litigation with those of patients who had completed litigation. Active litigants reported more pain than did postlitigants, but no differences were found in function or employment status. Patients with more pain and more objective findings were more likely to file claims. There was no evidence for improvement after litigation was settled. In fact, after settlement, 39% of patients improved, 55% showed no change, and 5% got worse. Sapir and Gorup[24] compared the results of radiofrequency neurotomy (RFN) for facet joint pain in litigants and nonlitigants; both groups did equally well.

In 1956, Gotten[26] reported that, of the 100 patients from an initial group of 212 patients with neck pain following whiplash whom he was able to locate years later, 88% improved after settlement. However, this study had only 40% follow-up and reported a high prevalence of chronic pain, despite litigation settlements. In 1961, Miller[27] concluded that whiplash-associated disorders in litigant patients was caused by "accident neurosis" and implied that patients are "cured by verdict," despite lacking data to support this conclusion.

Several studies have shown correlation between retaining an attorney and longer time to claim closure.[14,28,29] Consulting a lawyer was associated with a lesser chance of early claim settlement, longer treatment, and slightly worse function, but not with improvement in pain or return to work at 12 months.[28] In a no-fault, no-tort system, there is a shorter time to case closure.[29] However, some feel that time to case closure cannot be equated to improvements in pain and disability.[30]

Clinical Symptoms

Acute Versus Chronic Whiplash Pain

Acute neck pain occurs soon after injury. Chronic pain is defined not by an arbitrary determination of time but as pain that persists beyond the expected resolution of the structural injury. Because most patients with whiplash have recovered by 3 to 6 months, it is appropriate to define chronic whiplash as neck pain that persists beyond 6 months.

Neck Pain

Neck pain is the predominant symptom of whiplash injury. It may be midline or occur on either side or both sides. Pain is commonly referred to the trapezius muscle, shoulder, interscapular area, or arm, and occasionally to the face.

Arm Pain

Arm pain is common in chronic whiplash although, in the absence of neural compression, neck pain usually is more severe. Arm pain may be caused by radiculopathy as a result of neural compression from disk herniation or from preexisting foraminal stenosis rendered symptomatic by the trauma. In true radiculopathy, arm pain follows a dermatomal distribution, and there is objective neurologic deficit. More often, however, no objective neurologic deficit exists, despite arm pain caused by neural compression. By definition, this is not true radiculopathy; it might better be termed radicular pain. A lesion, such as lateral disk herniation or spinal stenosis of the lateral canal, is generally visible on magnetic resonance imaging (MRI). In the absence of neural compression, pain may be referred to the arm from a discogenic source[31,32] (eg, painful disrupted disk, midline herniation) or, less commonly, from a facet joint.[33,34]

Other sources of arm pain include shoulder pathology, ulnar nerve injury and/or entrapment at the elbow, and conditions unrelated to the neck (eg, brachial plexus lesion). Pain in the shoulder may be referred from the neck or caused by a primary shoulder problem.[33,35,36] Facet joint–mediated arm pain is usually in the shoulder or uppermost arm.[33,34] In a retrospective review of 34 patients with chronic neck pain located near the superomedial aspect of the scapula, Gorski and Schwartz[35] found that 24 patients (71%) had been in a prior MVC. All patients had restriction of cervical range of motion, a positive impingement sign, pain relief after subacromial injection with local anesthetic and corticosteroid injection, and abnormal shoulder radiographs. Chauhan et al[36] described a 22% prevalence of shoulder problems, most commonly impingement syndrome, in 524 patients with chronic whiplash injury. Carpal tunnel syndrome has been associated with shoulder and arm pain after whiplash.[37] Ames[37] postulated that such injury is the result of blunt trauma to the median nerve (ie, median neuropathy) from the steering wheel or dashboard rather than being true carpal tunnel syndrome.

Headache

Headache is the second most common symptom of whiplash. Cervicogenic headache, which may vary in severity and frequency, may be confused with migraine or tension-type headache. Cervicogenic headache almost always involves the base of the skull and frequently radiates to the crown of the head and frontal regions. It is often unilateral, and the side may vary in the same patient. This type of headache is often precipitated by prolonged static neck positions or repeated end-range flexion, extension, or axial rotation. There are several documented sources for chronic cervicogenic headache, including discogenic pain from C2-C3 and C3-C4, upper cervical disk herniation, an upper cervical facet joint, and the atlanto-occipital joint.[38-40]

Whiplash-Associated Disorders

Other symptoms associated with whiplash are referred to as whiplash-associated disorders. They include low back pain (LBP), visual disturbances, dizziness, tinnitus, weakness, fatigue, poor concentration or poor memory, difficulty sleeping, and secondary psychological changes (eg, depression).[41-43]

Low Back Pain

Cassidy et al[42] stated, "Low back pain is a common traffic injury with a prolonged recovery." Of 8,124 whiplash claimants, 4,473 initially had LBP. It was still present at 6 months in 30% to 42% of patients. Berglund et al[43] noted a 20% prevalence of chronic LBP 7 years after MVC. Barnsley et al[41] reported an approximate 40% prevalence of chronic LBP. The structural causes of LBP after MVC have not been studied specifically, but they do not appear to be different from the usual causes of chronic LBP. Sacroiliac joint pain, facet joint pain, and discogenic pain can occur after MVC trauma.[44]

Psychological Disorders

Peebles et al[45] compared psychological findings in patients with chronic whiplash with those in patients who had other chronic musculoskeletal pain. The authors found no differences with respect to the prevalence or types of psychological problems. They reported no characteristic psychological whiplash profile. Mayou et al[46] followed a group of patients for 1 year following MVC to evaluate early and late psychological consequences. Initially, almost 20% of patients suffered acute stress syndrome. At 1-year follow-up, 5% of patients met criteria for posttraumatic stress disorder, 18% had travel anxiety, and 12% had a mood disorder.

Psychological disorders secondary to pain and impairment would be expected to improve when pain is effec-

tively treated. RFN was used to treat 17 patients with both cervical facet joint pain and psychological abnormalities. All of the patients whose pain improved after RFN showed improvement in previously abnormal psychological tests scores.[47] In all but one of those who did not improve after RFN, the psychological abnormalities did not change. Furthermore, when pain recurred, psychological abnormalities soon followed. When pain responded again to RFN, psychological tests improved once again.

Fear avoidance, in which patients avoid activities because they are fearful of those activities, is a psychological problem that is important in creating or perpetuating impairment.[22] Common fears include fear that activity will worsen pain, that increased pain indicates further structural damage, and that activity will cause reinjury. Some patients fear that persistent pain is an indication of more serious pathology. In some instances, patient function increases after the patient is reassured by the physician that it is fine to be active despite pain and that the patient will not cause further damage to himself or herself.

Structural Etiology of Chronic Neck Pain Caused by Whiplash

There are no findings on history or physical examination that are specific to any one structural cause of chronic axial neck pain. However, the history and physical examination are extremely important in excluding serious medical problems or causes of pain other than neck pain. The history provides information regarding the mechanism of injury, severity of pain, degree of impairment, and patient values, all of which contribute to evaluation and treatment.

The facet joints are the most common source of chronic neck pain after whiplash injury.[48-50] Some patients have pain that arises from a disk, and some have a combination of facet joint pain and discogenic pain.[49,50] The patient with acute whiplash may have pain secondary to injury to muscles and ligaments. However, as in most soft-tissue injuries, muscle and ligament injury heals in 6 to 8 weeks. There is no evidence that chronic neck pain is the result of whiplash affecting a chronically strained muscle or a chronically sprained ligament.[12]

Facet Joints

Biomechanical and autopsy studies indicate that the facet joints can be damaged in MVC.[7,8,51,52] Clinical prospective studies provide level I evidence that the facet joints are the most common source of chronic pain resulting from whiplash injury.[48,49] Chronic neck pain can be traced to a facet joint in 49% to 54% of patients with whiplash injury who have neck pain only and in 60% of those with both neck pain and headache.[48-50] In patients with cervicogenic headache following whiplash injury, there is a 53% prevalence of C2-C3 facet joint pain.[48]

Facet joint pain can be diagnosed only by anesthetizing the putative painful joint.[48] Cervical facet joints are innervated by the medial branches of the cervical dorsal rami. A single block is not definitive because of the high false-positive rate. The diagnosis is made when pain is relieved on two separate occasions after the joint has been anesthetized by anesthetic injection of the medial branches of the cervical dorsal rami serving the putative painful facet joint (ie, medial branch block).

Discogenic Pain

The data implicating the disk as a source of chronic neck pain after whiplash are not as clear as those implicating the facet joints. Anatomic, biomechanical, and autopsy studies indicate that the intervertebral disks are innervated and that disks can be injured during whiplash.[8,53] In addition, uncontrolled clinical studies are consistent with observations in healthy volunteers that cervical disks are a potential source of pain[31,32] and that anterior cervical diskectomy and fusion (ACDF) relieves pain in approximately 70% of well-selected patients.[54,55]

Diskography has been used to identify painful cervical disks.[31,32,56] This may be a useful test when strict protocols are observed and the results are interpreted in conjunction with the other clinical information. Diskography should be reserved for the patient with refractory pain who is being considered for surgery; it should not be used simply to establish the diagnosis. Diskography may reveal multiple painful disks and thus eliminate surgery as a consideration.[56]

There is some evidence that cervical disk injection may precipitate pain from a facet joint and that some patients may have both disk and facet joint pain.[35] To maximize the chances of a true-positive diskogram, a medial branch block at the index level should be performed before diskography and be negative. In addition, injections of disks adjacent to the painful disk or disks should be painless.

Craniocervical Ligaments

Radiographic studies provide evidence that alar and transverse ligaments may be damaged in whiplash.[57,58] A series of MRI studies[57,58] demonstrated that ligamentous injury was more common in patients with a history of whiplash than in asymptomatic individuals. These studies did not correlate imaging findings with patient symptoms. When these lesions are indeed symptomatic, their location at the C1 level might render them a potential source of cervicogenic headache but not necessarily of neck pain. However, these ligamentous injuries were not found in cervical spine specimens subjected to low-impact simulation.[59]

Other Soft-tissue Injury

Soft-tissue injury may be the most controversial and misunderstood clinical aspect of whiplash injury. This is partly because soft-tissue injury is a nonspecific and potentially confusing term that has little clinical value. Soft-tissue injury implies only that some part of the cervical support structures other than bone has been injured. Using this definition, injury to cartilage, facet joints, and disks all would be considered soft-tissue injury. There is no evidence that neck pain that has lasted for >3 to 4 months can be attributed to chronic strain or sprain, although these terms are used frequently, especially in the medicolegal context.[12] Furthermore, no studies adequately demonstrate that damage to the soft tissues alone is a primary cause of moderate to severe chronic neck pain. Muscles may become sore from chronic poor posture; however, this pain is usually described as a mild to moderate ache and is not sufficiently severe to cause meaningful impairment.

Treatment

Acute Neck Pain

Once serious injury is ruled out by clinical examination and, if necessary, cervical radiographs, initial treatment should consist of explanation, education, and reassurance. In the absence of significant neurologic abnormalities or suspicion of fracture, advanced imaging studies usually are not necessary for at least 3 months. The physician should recommend that the patient remain active, despite pain, and explain the generally favorable natural history. This cannot be overemphasized. It is not clear that any early intervention can prevent chronic symptoms.[60] Kongsted et al[60] performed a prospective randomized study comparing the outcomes at 1 year for acute whiplash patients treated with immobilization in a cervical orthosis, active mobilization using range-of-motion exercises, or recommendations to "act as usual." At 1 year, there were no significant differences observed among the groups except for a higher rate of disability in the cervical orthosis group. At 1 year overall, 48% of the patients reported considerable neck pain, 53% had some degree of disability, and 14% were still off work.

That said, it appears that patients who remain active despite pain generally have a more favorable outcome than do those who rest excessively or avoid activity.[61,62] Exercises should be prescribed rather than just suggested. It is not usually necessary to refer a patient to a physical therapist in the acute stage. There are many informational booklets and books available.[63] The evidence regarding spinal manipulative therapy (SMT) is conflicting.[64] Several reviews have found no evidence that SMT is useful when used alone or in conjunction with passive modalities.[64] However, Hoving et al[65] reported slightly better outcomes when SMT was combined with exercise.

Vassiliou et al[66] compared the outcomes at 6 weeks and 6 months in a randomized trial that compared cervical orthosis with 10 physical therapy visits. The authors concluded that physical therapy that includes active exercise is superior to treatment with a soft collar. However, at the same time, it must be noted that patients who utilized more health care in the first 30 days after injury had slower recovery than did those who used less care.[67]

Chronic Neck Pain

Rehabilitation

Physical therapy is usually the first treatment prescribed for the patient with chronic neck pain. Effective rehabilitation requires strengthening exercises and training in body mechanics. Modalities and stretching are often used, as well, despite little or no evidence of efficacy. Exercise alone is rarely curative. On average, studies report between 25% to 75% reduction in pain.[68] The best available evidence supports exercise as effective treatment for chronic neck pain,[69,70] but there is just one study[68] that is specific for patients with neck pain resulting from whiplash. The authors compared patients treated with advice alone to those treated with advice plus 12 sessions of exercise over 6 weeks. Exercise plus advice produced somewhat superior results, with small improvements in pain intensity and bothersomeness, disability, and quality of life after 6 weeks. However, the benefits were not apparent at 12 months. Patients with higher levels of pain and disability improved to a greater extent.[68]

There do not appear to be meaningful differences between whiplash patients and others with chronic neck pain.[71] Therefore, the outcome data of rehabilitation for chronic neck pain should be applicable to whiplash patients.

There is fair evidence that exercise directed toward strengthening the neck and shoulder-thoracic area can reduce pain and improve function in the patient with chronic neck pain.[69,70,72-74] The patient must continue to exercise to maintain these gains. Strength training and endurance training both have been shown to be better than stretching and aerobic training.[72] Intensive exercises are more effective than light exercises[73] but not necessarily more effective than ordinary activity.[74] There is moderate evidence that a multidisciplinary program can reduce pain, improve range of motion, improve function, and decrease disability compared with placebo therapy.[75,76]

Rehabilitation prescriptions must be specific and directed toward strengthening the muscles that are usually weak—the anterior muscle group, the interscapular muscles, and the posterior neck muscles. Also, there must be a specific prescription for body mechanics training for activities of daily living, with special attention given to work and

recreational ergonomics. In addition, good rehabilitation indirectly provides behavior modification to help overcome fear avoidance with respect to activities of daily living and exercise without supervision.

Injections

Level I evidence supports the efficacy of RFN as a treatment for chronic neck pain that arises from facet joints.[77,78] RFN is appropriate only for the patient with proven facet joint pain. RFN is the coagulation of the medial branches of the dorsal rami that are responsible for conducting pain from the joint. The indication for RFN is significant pain relief following controlled anesthetic blocks of the medial branches of the nerve supply to a specific facet joint. Complete pain relief can be achieved in many patients with pure facet joint pain and in 86% of patients with facet joint–mediated cervicogenic headache.[77-79] Other patients will experience ≥50% relief, which is sufficient to make a meaningful difference in function and quality of life. Some of these patients may have other pain generators in addition to the facet joints. Relief lasts a median of 270 to 400 days, after which pain recurs because the nerves regenerate. Repeat RFN is usually effective in relieving recurrent pain.[78] Intra-articular injections of corticosteroids do not provide long-term relief; however, they may provide short-term relief, during which rehabilitation may be more easily performed. There are no data to support the use of trigger point injection.

There is no evidence to suggest that cervical epidural corticosteroid injection is effective in relieving axial neck pain. The data for radicular pain are mixed.[80-82] At best, 30% of patients have partial but long-lasting relief and another 30% have complete relief with transforaminal injection.[80-82] However, it has been suggested that cervical epidural corticosteroid injections are not useful for traumatically induced radicular pain.[82]

Case Example

A 46-year-old man was the fully restrained driver of a car struck from the rear by a sport utility vehicle moving at 25 mph. The patient had immediate neck pain, which persisted for 9 months and significantly interfered with his quality of life. Pain was located in the right side of his neck at C5-C6 and radiated to the right trapezius and proximal interscapular region. Physical examination revealed only guarded range of motion. MRI showed a very small midline disk herniation at C5-C6. The patient failed to improve despite physical therapy, medication, and epidural corticosteroid injection. Medial branch blocks were performed to denervate C5-C6 and C6-C7. After the local anesthetic phase, the patient experienced 80% relief of pain for 3 to 4 hours. A second injection 2 weeks later yielded the same results. RFN was performed to denervate these two levels. The patient was worse for 1 week, then was markedly improved. Symptoms recurred at 11 months. A repeat RFN was performed and was equally successful in providing pain relief.

Medication

Medication may play a role in the treatment of neck pain after an MVC.[83] For the patient with acute neck pain, the most useful drugs are nonsteroidal anti-inflammatory drugs (NSAIDs), opioid analgesics, and muscle relaxants (which may be useful for up to 14 days after the accident).[83]

The response to NSAIDs for treating chronic neck pain is unpredictable. It is appropriate to try several NSAIDs for up to 2 weeks each in patients with low risk for systemic side effects. When there is a meaningful response, the drug should be continued; when there is no response to three trials of different NSAIDs, further NSAID trials are not likely to be helpful.

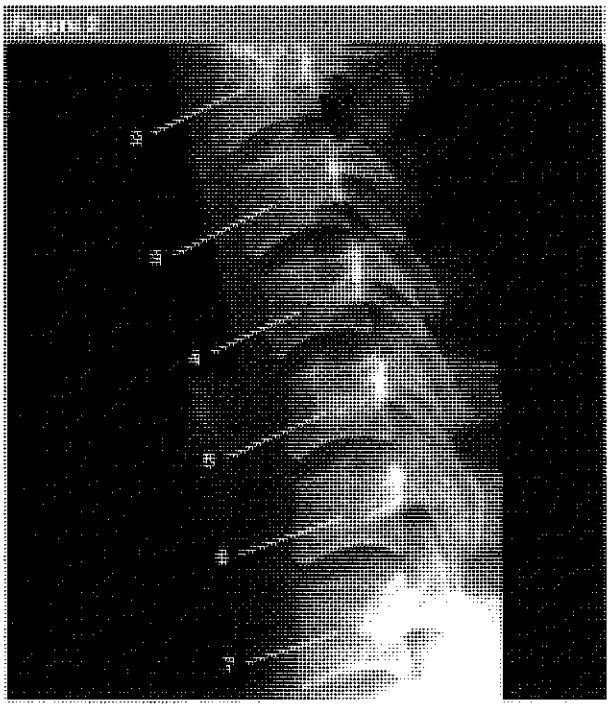
Opioid analgesics can play a valuable role in well-selected patients with chronic, moderate to severe neck pain that has proved to be refractory to other treatments. Although there are no data specific for treating chronic neck pain, opioids have proved to be valuable in the treatment of LBP and other chronic musculoskeletal conditions. Tricyclic antidepressants (eg, nortriptyline) have been shown to be useful in chronic axial LBP, but there are no data specific to chronic neck pain. Anticonvulsants (eg, gabapentin, pregabalin, topiramate) may be helpful in neuropathic pain but rarely provide benefit to the patient with axial neck pain.

Spinal Manipulative Therapy

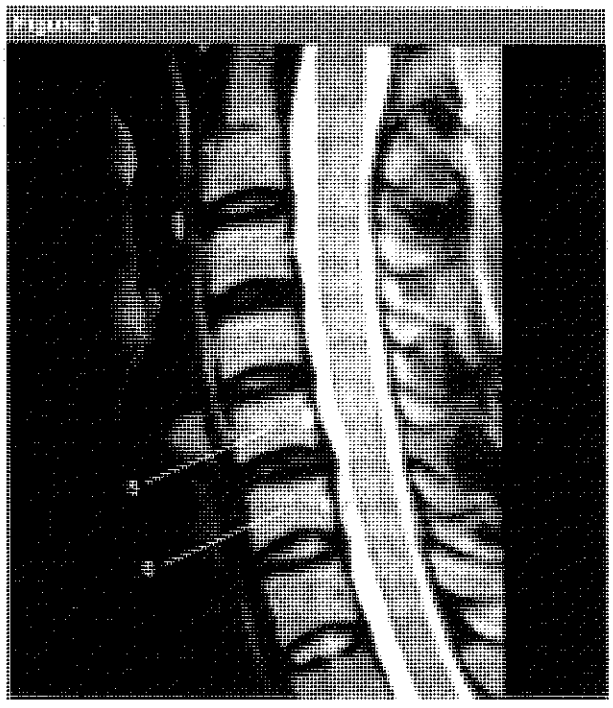
Although there is no consensus about the efficacy of SMT for chronic neck pain, it remains one of the most popular treatments.[64,65] When SMT alone was compared with exercise alone and exercise plus SMT, both exercise groups did somewhat better than did the group with SMT alone, although all treatment groups improved. However, when intensive training, physiotherapy, and SMT were compared, there were no differences between groups, and all groups improved. Although there are anecdotes about serious complications from SMT, the incidence is quite low.

Cervical Orthoses

Cervical orthoses are of no value in the patient with acute whiplash injury. Persson et al[84] compared three groups of patients with long-lasting radiculopathy who were treated with surgery, physical therapy, or a cervical collar. No significant clinical or statistical differences were found between groups at 12-month follow-up; all groups did reasonably well. However, these results are blurred by a high number of patients who crossed over between groups.



Lateral radiograph of the cervical spine in a 39-year-old woman showing mild disk degenerative changes at C5-6 and reversal of the cervical lordosis.



Same patient as in Figure 2. Sagittal MRI scan showing narrow disk with mild dessication and small herniation at C5-6.

Surgery

There is controversy regarding the role of surgery for the patient with axial neck pain. Garvey et al[54] reported on 87 patients, including 25 with chronic whiplash injury, who underwent ACDF for axial neck pain. At 4-year follow-up, 83% of patients had good to excellent results and statistically significant improvements in both the Oswestry Disability Index and the modified Roland Morris Disability Questionnaire. Palit et al[55] reported on 38 patients who underwent ACDF for axial neck pain. The authors noted significant improvement in pain and Oswestry Disability Index score; 79% of patients were satisfied with their outcomes. Similar findings were reported for ACDF at C2-C3 and C3-C4 for discogenic cervical headaches in a study with nine patients.[38]

Surgery may be considered for the patient with severe pain, significant impairment, and good psychological health who has not improved sufficiently with high-quality nonsurgical care. The surgeon should order plain radiographs with flexion and extension views (to screen for instability and evaluate levels adjacent to the index level for degenerative changes) and MRI. Although somewhat controversial, diskography might be considered. Diskography can be useful when it is interpreted in the context of the history, physical examination, and other tests. To interpret diskography, the following two conditions must be met: (1) no significant pain relief after a medial branch block, combined with disk injection demonstrating concordant pain reproduction at the index level or levels, and (2) no pain at adjacent levels. In the patient requiring surgery for axial neck pain, better overall surgical results were achieved when diskography was included in the preoperative evaluation than when it was not used.[85] Because ACDF eliminates motion at the painful segment, theoretically, if there were both a painful disk and facet joint, ACDF would treat both.

Case Example

A 39-year-old woman with no preexisting history of neck pain was injured in a rear-end MVC. When seen 1 year after the accident, she had predominant axial neck pain and restricted range of motion. The neurologic examination was normal. She did not respond to treatments that included physical therapy, medications, and medial branch blocks. Plain radiographs showed mild degenerative changes at C5-6 and reversal of the cervical lordosis (Figure 2). MRI showed a disk herniation at C5-6 (Figure 3). Diskography was positive (ie, concordant pain) at C5-6 and negative at adjacent levels. The

patient underwent ACDF with allograft bone and plate fixation at C5-C6. Her cervical pain and headaches resolved by 2 months postoperatively, and the patient returned to full activity at 3 months. Her fusion healed without complication.

Summary

For the patient with chronic neck pain caused by whiplash, a careful history and examination are necessary to establish or exclude neurologic deficits, nonspinal musculoskeletal causes of pain, and systemic illness. The most likely sources of mechanical neck pain are facet joints, intervertebral disks, or both. The patient should be referred for physical therapy to strengthen the neck and upper back muscles, learn body mechanics and ergonomics, and overcome fear-based avoidance of activity.

When there is no meaningful improvement with physical therapy, the facet joints should be investigated by medial branch blocks. The levels tested are selected based on the location and referral patterns of the pain, the location of tenderness on physical examination, and the location of any abnormal segments on MRI or plain radiographs.

If medial branch blocks provide significant relief of pain on two separate occasions, RFN should be considered. When medial branch blocks determine that the facets are not pain generators, and pain is severe, the disks are the most likely pain generator, and surgery may be an option. When surgery is not an option, long-term medical management, often involving opioid analgesics, may be considered.

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Evidence-based Medicine: There are 25 level I/II studies: 12 prospective randomized controlled trials (references 60-62, 65, 66, 68, 69, 72-74, 77, and 84), 9 prospective prognostic studies (references 3, 18, 21, 23, 28, 42, 44, 48, and 71), and 4 systematic reviews of randomized controlled trials (references 14, 15, 64, and 70). There are 12 biomechanical or anatomic studies. The remaining references are level III/IV (observational studies, case-control reports, or cohort studies) or level V (expert opinion).

Citation numbers printed in **bold type** indicate references published within the past 5 years.

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Understanding and Managing the Impact of Vehicle Crashes: Reconstructing the "Accident"

- A car crash may be an "accident" but the way in which forces are transferred to the occupants and their resulting injuries are not accidental - they are defined by the laws of physics and mechanics.
- Research into the biomechanics of injury and the incorporation of innovations such as frontal and side airbags, lap and shoulder belts, and booster seats for children have reduced injury and death from motor vehicle crashes.
- Today, however, significant numbers of people still suffer neck "whiplash" injuries in rear end impacts and serious head, chest, thigh, leg, foot and pelvic injuries in side "T-bone" crashes.
- Discoveries in the Department of Orthopaedics and Sports Medicine are defining the mechanics of these collisions and enabling improvements in vehicle safety equipment.

Despite significant improvements in vehicle safety equipment, including frontal and side airbags, significant numbers of people are still injured or killed every year in auto crashes. The new safety equipment was designed to meet federal regulations based on testing with crash dummies, usually with 'standard' hypothetical types of impact, such as a straight on crash into a wall. As such, they do not address such real world problems as out-of-normal driving positions of occupants and non-standard directions of crashes. The faculty of the Department of Orthopaedics and Sports Medicine have collaborated with the Harborview Injury Research and Prevention Center in a unique program that enables us to determine the actual injuries in real-world crashes. With the consent of the individuals involved, we document the injuries and investigate the specific direction and speed of the crash, the function and use of safety restraints, and the points of occupant contact in the vehicle to determine the mechanics of the injuries sustained. This information, combined with laboratory studies, has provided valuable insights into injury prevention. The following are short summaries of some of the findings.

Femur fractures in head on collisions

Just prior to a frontal collision, the vehicle and occupant are traveling at the same speed. At impact, the vehicle slows very rapidly, but the occupant continues forward at the original speed until encountering the restraint devices. The restraining devices, shown in Figure 1, include the airbag to stop the forward motion of the head, the shoulder belt to stop the torso, the lap belt to stop the pelvis, and the knee bolster to stop the legs. This system attempts to apply forces across the whole body so that the occupant "rides down" the collision. The knee bolster is the padding on the dashboard that absorbs impact forces as the occupant's knees move forward. These have been designed based on biomechanical impact testing of isolated femurs.

However, in a series of crashes, we were perplexed to find that normal adult occupants were sustaining femur fractures at crash speeds well below that at which femur fracture would be expected. To solve this mystery, we constructed computer models of each injury to determine the approximate forces acting. None of the occupants studied had any significant osteoporosis or other bone disease that might indicate weaker than normal bone that would predispose them to femur fracture. Almost all had been drivers, and all were in vehicles that had braked hard prior to impact, as indicated by skid marks from their tires. The forces we calculated did not reach levels expected to create femur fractures, yet each occupant had a documented femur fracture. We then postulated

Figure 1: A schematic diagram showing the forces acting on an occupant during "ride down" of a frontal collision. Force is required to slow the occupant's motion inside the vehicle, and is distributed between; head and airbag, chest and shoulder belt, pelvis and lap belt, and knees and dashboard. The external force on the knee during dashboard contact causes an axial compressive force acting on the femur, which is increased by internal muscles forces due to tensing the leg during braking. Dashboard stiffness has been designed based on the strength of the femur. Muscle contraction adds considerable compressive force to the external force from dashboard contact, resulting in occupants fracturing their femurs at lower than expected impact speeds. Therefore dashboards should be designed to be less stiff to accommodate the combined compressive force from external contact and internal force from muscle contraction.

that additional forces along the axis of the femur could have been applied by the occupant contracting muscles along the leg while bracing during the crash. When the maximum contractile forces applied were estimated based on muscle mass, and added to the calculated force of impact of the femur into the dashboard, the forces exceeded the average values necessary to create a fracture. We concluded that dashboard impact absorption systems, which are based solely on the external forces applied to the femur during contact, do not replicate the scenario in an actual frontal crash where large internal forces may also be applied by muscle contraction. Therefore, we are proposing that dashboard "knee bolsters" may be too stiff as currently designed and may need to be softened in consideration of the internal muscle loads that are associated with these crashes.

Chest and pelvic injuries in side impact (T-bone") collisions

In a side impact ("T-bone") collision, usually at an intersection when one vehicle runs a stoplight, the impacting vehicle hits the door of the struck vehicle, and deforms it inwards. (Alternatively, in a single vehicle crash, the car misses a curve, slides off the road, and contacts a tree with its door.) As the door bows inward, the inner surface of the door contacts the occupant, causing direct contact injuries and pushing the occupant towards the center of the car. In some cases, when the vehicle has a large center console, the occupant may be crushed against it, see Figure 2, causing additional injury. Recently, curtain airbags have become available which significantly protect the head from injury in these collisions, however other types of airbags such as those designed to protect the thorax, have been less effective. This is particularly the case when the occupant driver, sensing an approaching vehicle from the left, attempts to swerve to the right, which causes the driver to be thrown against the driver's side door, just as the airbag installed in the door deploys, which could result in airbag-induced rib fractures.

In reconstructing a series of side impact collisions, we discovered that the most significant factor related to the severity of side impact injuries to the chest and pelvis was the amount of intrusion of the door during the impact. (This is related to the weight and speed of the striking vehicle and the location of contact on the door). Further, in cars with center consoles, injuries were generally more severe. Therefore we are proposing a system that attempts to move the driver away from the incoming door during the impact. The elements of the system, shown in Figure 3, include a crushable center console, a seat with a track that allows it to move sideways towards the center of the car, and a stronger seat structure. In this system, the door hits the side of the seat, not the side of the occupant, and pushes the seat and occupant away from the incoming door into the center console. The console itself is soft enough to be crushed, absorbing the energy that otherwise would case injury. Our ultimate goal is to minimize the risk of serious injury from side impact by optimally managing the full range of forces that might be countered. A patent for this system is pending.

Whiplash injuries of the neck from rear impact

The most common injury in car cashes is "whiplash", which causes damage to the soft tissues of the neck from a low speed rear impact. The impact to the car from behind causes the occupant's body, Figure 4, to be thrust forward. Unless it is in firm contact with head rest, the head does not accompany the body in its forward thrust. Shear and extension loads are applied to the neck, resulting in unnatural distortion of the lower part of the cervical spine. The head then impacts the head restraint that limits the head's rearward motion. However, most head restraints are very elastic and during contact they bend backward, then propel the head forward like bouncing off a trampoline.

Figure 2: Side impact crash in which occupant was trapped between incoming door and the center console and sustained severe pelvic fracture.

An instant later, the forward motion of the torso is stopped by the shoulder belt, but the head continues to move forward, again inducing a horizontal or shearing force in the neck. Finally, as the head continues forward, it bends the neck into forward flexion. In our laboratory we found that these horizontal translations between adjacent vertebrae may be an important mechanism by which the soft tissues of the neck can be injured in a whiplash event.

Even though head restraints are required in all vehicles currently for sale, the incidence of whiplash injury has not diminished. By studying over 400 actual rear end impacts and the seated positions of over 700 drivers, we discovered that in the majority of cases head restraints are not adjusted properly. They are generally set too low and are too far behind the head. We are working to define ways in which the head restraints can be ideally positioned for each individual. We have designed and tested an improved head restraint that allows the user the ability to set its position both vertically and horizontally relative to the head, and that deforms during impact, catches the head, and eliminates most of the forward rebound. This design significantly reduces head to neck motions during rear impact. In the future the frequency of whiplash may be reduced by the application of such designs that protect the neck by keeping the head aligned with the body.

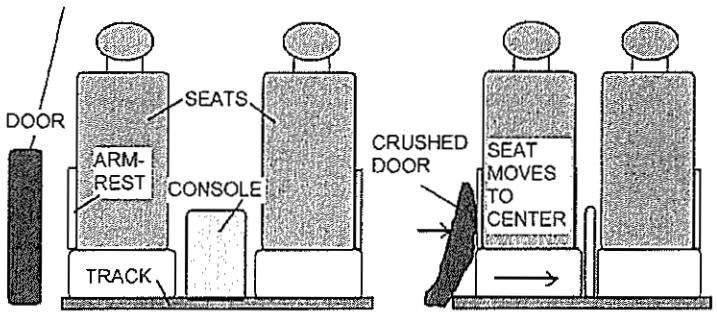

Figure 3: (left) schematic diagram of a (US patent pending) system to prevent or reduce severity of such injuries, the SEATS are on a lateral TRACK with a crushable center CONSOLE and a guard/ARMREST on the door side of the seat, (right) during a side impact, the crushed door impacts the seat and armrest/guard, displaces the seat and occupant towards the center, crushing the center console and moving the occupant away from the door. (Another modification would be that the seatbelts would be attached to the seat).

Conclusion

Investigation of the mechanics of how real occupants respond to the forces acting in real collisions (as opposed to studies limited to crash dummies) provides valuable new insights into the mechanisms of vehicular injuries. These insights will enable us to reduce the potential for occupant injuries in car "accidents". Our team will continue to study the real world of accidents in pursuit of further advances in the protection of the driver and passengers. We have a particular interest in reducing the risk of injuries to children in the rear seat during side impact collisions, recognizing that side impact airbags are designed and positioned for adults.

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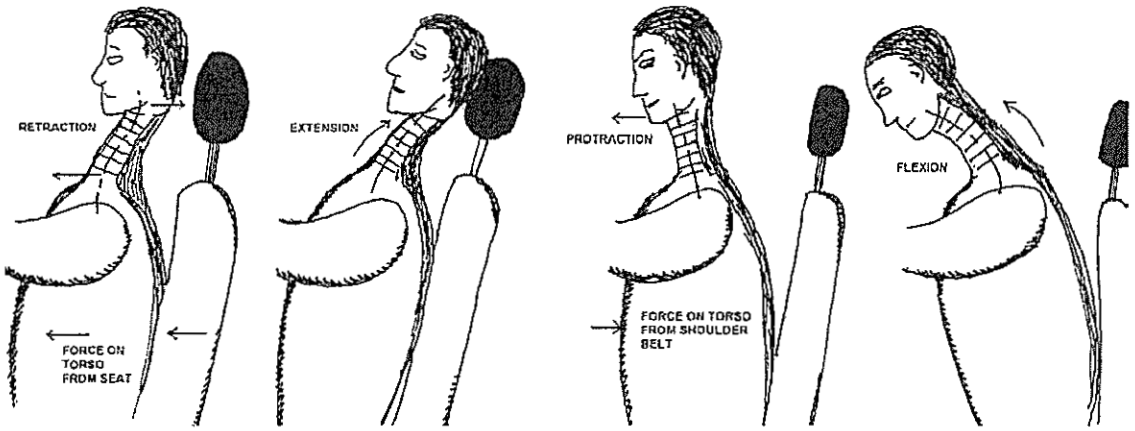

Figure 4: (upper) the four phases of "whiplash", left to right, torso is thrust by seat contact under head causing horizontal shearing or protraction of vertebrae, head falls back into head rest causing neck extension, torso stops due to shoulder belt acting on chest while head rebounds from head rest causing protraction or horizontal shearing, and finally, head continues forward causing neck flexion.

(lower) A device to reduce head to torso motion and neck forces, has a lower hinge to move head rest closer to head (reducing initial retraction), while upper hinge adjusts both a head cushion and neck cushion (not shown) to the occupant's posture, reducing extension. The hinges are "plastic" that is they move slightly and absorb energy from impact with the head and reduce head rebound, thus limiting protraction and flexion of the neck.

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The Response of Human Volunteers to Rear-End Impacts

The Effect of Head Restraint Properties

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Study Design. Human volunteers were subjected to a rear-end impact while sitting on a standard automobile seat, and sagittal plane kinematic responses were quantified. The effect of changing head restraint properties was determined by use of a repeated measures design.

Objective. To determine the forces acting, and relative motions resulting, on volunteers in a rear-end impact and the effect of head restraint properties.

Summary of Background Data. In several recent studies of the kinematics of the cervical spine during rear-end impact, a forward thrust to the lower cervical spine was produced, and a transient S shape of the spine resulted while the head remained upright during the initial phase of the impact. This may result in nonphysiologic intervertebral motions and tissue strains.

Methods. Nineteen automobile seats were first tested, and a modified head restraint was designed. Each volunteer sitting on a standard vehicle seat was subjected to an impact pulse of 3*g* with a 4-kph speed change. Testing was performed first with the modified head restraint, then again after replacement by the head restraint that came with the seat. Kinematic responses were compared for both head restraints by use of a repeated measures analysis of variance.

Results. There was a measurable time difference between peak chest and peak head accelerations, which resulted in the chest being thrust forward by the seat back before the head was thrust forward by the head restraint. The modified head restraint significantly reduced the contact time difference and therefore decreased the relative chest-to-head forward motion.

Conclusions. Volunteers seated on a standard automobile seat demonstrated differential sagittal plane motion between the chest and head. It is possible to significantly decrease the relative chest-to-head motion by altering the characteristics of the head restraint. [Key words: cervical spine whiplash, rear-end impact, biomechanics] **Spine 2001;26:2432–2442**

Whiplash injuries of the cervical spine resulting from rear-end motor vehicle accidents continue to be the single most expensive medical claim to insurers.[12] diagnosis of whiplash is confounded by a general lack of objective symptoms, but it may result in a long-lasting chronic condition.[1,4,5,26,27,32] In the United States, head restraints were introduced in vehicles in 1969, to limit rearward angular displacement of the occupant's head relative to the torso with the aim of preventing whiplash.[8] O'Neill et al[22] showed that 29% of drivers of vehicles without head restraints reported neck injuries after a rear-end impact, compared with 24% who had vehicles with head restraints. A significant number of drivers still reported injuries, which was attributed partly to the fact that many of the head restraints were not properly adjusted.[22] In a recent survey of the seated positions of 719 drivers, 25% were found to have the head restraint adjusted below ear level, and 45% had head restraints that were more than 7.6 cm behind the back of the head.[38] The Insurance Institute for Highway Safety found that only 5% of model year 1995 automobiles had head restraints that allowed positioning above ear level and closer than about 7.6 cm to the back of the head.[12] Therefore, in many cases this safety device may not be effective in controlling head and neck motion during rear-end impact.

The response of an occupant to a rear-end impact has been well documented.[17–20,23,29,34–36,38] The seat is pushed toward the occupant. The occupant's torso contacts the seat back first and is thrust forward. The head falls backward toward the head restraint, resulting in cervical spine extension. At this point the vehicle impact is over. The torso continues to move forward, and the head rebounds forward off the head restraint at a later time. Several recent studies, most performed using cadaveric cervical spine preparations, have identified an initial transient S shape of the cervical spine in response to a rear-end impact, as a result of the lower cervical spine being thrust forward while the head remains initially level. This S shape has been associated with nonphysiologic extension of the lower cervical segments,[24] abnormal interactions of the facets,[23,40] facet capsular tissue strains,[6,30] and transient compression of the neural tissues.[7,28] Although it is not possible to identify which of these nonphysiologic events actually results in whiplash pain, all of these studies consistently described the same S-shaped transient motion of the cervical spine resulting from the lower cervical segments being thrust forward, causing the lower cervical spine to extend while the upper cervical spine flexes as the head remains transiently upright, before the head and spine start to extend. In the studies described above, these motions occurred because a force was applied to the sled apparatus to which the lower cervical spine was attached, while the

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head had no head restraint. In the present study, the first goal was to determine whether motions corresponding to those observed in cadaveric studies, specifically the chest moving forward of the head during the initial phase of the impact, occur also in human volunteers sitting on a typical automobile seat subjected to a rear-end impact.

The effect of the seat in modifying occupant motion in rear-end collisions has been recognized for some time.[2,21,25,31] The recommendations for resisting higher speed rear-end impacts have included increasing the height of the seat back, contouring the upper third of the seat, and increasing seat rigidity and plastic deformation to absorb energy in rear-end impacts. Two approaches to improving seat performance in low-speed rear-end accidents have been recently demonstrated. Lundell et al[15] described the WHIPS seat, which allows controlled reclining of the seat back during rear-end impact. This was found to reduce the energy returned to the torso after impact to between 20% and 30%, compared with between 30% and 45% for a standard seat. Alternatively, Wiklund and Larsson[44] described the SAHR seat, which has an active head restraint design. A plate in the seat is activated by the load of the torso acting against the seat, forcing the head restraint upward and forward during impact. This limits head rearward rotation and neck extension. In these studies, the chest-to-head differential motions were not specifically considered. Therefore, the second goal of the present study was to determine whether chest-to-head differential motions could be reduced by altering the properties of the head restraint.

These two goals were accomplished by the use of human volunteers seated on a typical automobile seat. The seat was chosen from previous testing of several vehicle seats to be reasonably representative of most seats. The chest and head kinematics of each volunteer in the sagittal plane were tracked by accelerometers. A repeated measures analysis was used to compare peak accelerations, time difference between chest and head peak accelerations, and relative chest-to-head motions with a standard head restraint or a modified head restraint in place on the same seat. The modified head restraint, shown in Figure 1, was designed to provide a higher frame, allow forward adjustment, have a stiffer connection to the seat back, and have an air cushion to conform to the shape of the volunteer's back of head, neck, and torso.

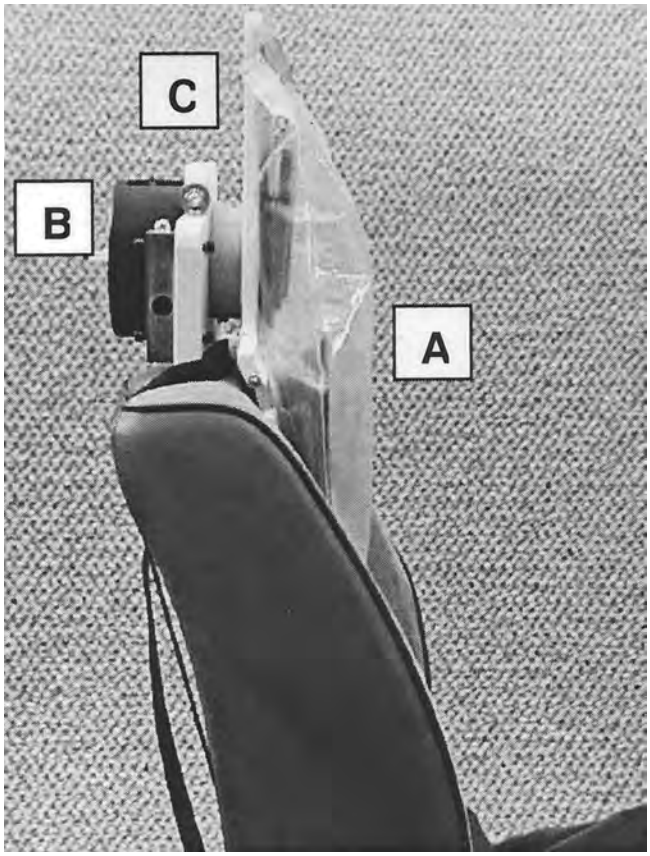

Figure 1. Prototype retrofit head restraint mounts on posts of standard head restraint; couples to a front plate on the seat back to stiffen the head restraint to seat back connection; has an inflated air cushion (A) to contour to the occupant's head neck and torso, with a discharge valve (B) to reduce rebound from the seat; and is higher and allows forward adjustment (C).

■ Materials and Methods

Basic Hypotheses and Study Design. The first hypothesis tested was that human volunteers seated on a standard seat would respond to rear-end impact force with differential motions between the head and torso. Specifically, if the peak force on the torso from the seat back occurs before the peak force acting on the head from the head restraint, the torso and the lower cervical spine will be thrust forward before the head. The second hypothesis was that altering head restraint geometry can alter the occupant's kinematics, resulting in a more uniform thrust against both the head and the torso. To test the hypotheses, 19 automobile seats were first tested for their response to impact against the head restraint and seat back, and a modified head restraint was developed. Then a total of 28 volunteers were subjected to a low-speed rear-end impact, first with the standard head restraint and then with the modified head restraint, and their kinematic responses were compared by use of a repeated measures design to test the hypotheses.

Seat and Head Restraint Testing and Development of a Modified Head Restraint. A mechanical tester, shown in Figure 2, was developed to determine the stiffness and energy return of the seat back and the head restraint. It consisted of a four-bar swinging pendulum with a 35-cm fall height using 90 kg of dead weights. The pendulum had a contact plate that impacted against the seat, or separately against the head restraint. Impact force was derived from accelerometer measurements and from the known mass of the pendulum. Seat back and head restraint angular displacements were measured by use of an inclinometer attached to the pendulum arm. The impact velocity of the pendulum against the seat was 11 kph, resulting in loading the seat at 2*g*, similar to the load anticipated in the volunteer study.

Testing was performed on 19 randomly selected vehicle seats. Six seats with double-post adjustable head restraints were tested with a standard head restraint and then with the modified head restraint, shown in Figure 1. Stiffness (peak impact force/peak angular displacement from point of contact) was calculated for the head restraint with respect to the seat

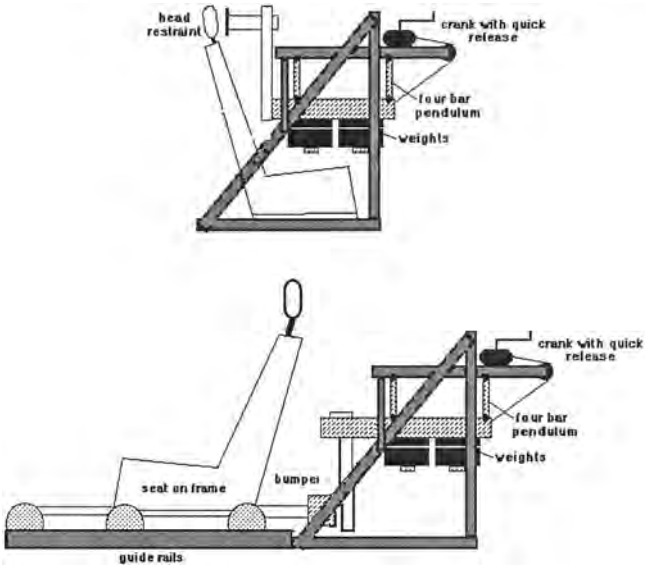

Figure 2. Schematic diagram of a mechanical tester for impact testing of seat backs and head restraints (above), with a four-bar pendulum, configured for impact to a seat on a wheeled frame with a bumper and guide rails (below).

back. Energy return was calculated as peak angle of rebound from point of contact of the seat or head restraint. Analysis of the data was performed by use of a Wilcoxon nonparametric ranked sign comparison to test for differences in head restraint stiffness and energy return of the seat with the standard head restraint and then with the retrofit antiwhiplash device in place.

A modified head restraint, shown in Figure 1, was designed on the basis of these principles:

Provide a surface that can self-adjust to the contours of the occupant's head, neck, and thoracic spine during impact to minimize differences in time of contact of the head with the head restraint and the torso with the seat back.

Provide an energy absorber to reduce rebound of the head from the head restraint and rebound of the torso from the seat back after impact.

Provide a stiffening frame to couple the head restraint connection with the seat back.

Ensure that the head restraint can be adjusted upward so that the center of gravity of the occupant's head is below the top of the head restraint, and forward so that it is as close as possible to the back of the head.

Testing of Human Volunteers with a Prototype Head Restraint Compared with a Standard Head Restraint. To test the hypotheses related to kinematics of occupants in vehicle seats subjected to a rear-end impact, 1 of the 19 seats previously tested was selected as representative of the properties of most vehicle seats. It was mounted on a six-wheeled frame running inside guide rails and had an impact-absorbing bumper constructed of two aluminum channels separated by two sets of rubber doughnuts. Energy was provided by the same pendulum used to test the seats in the previous study but with more weight added (total pendulum weight was about 73 kg). Its drop height produced a velocity of the pendulum at impact of about 6.4 kph (4 mph), and bumper compression resulted in an impact with an acceleration rising linearly to a peak of about 3*g* in about 80 msec, followed by a deceleration reaching a peak of −2*g* caused by frictional interaction of the wheels of the sled with the frame rails. Overall, the sled traveled about 40 cm because the impact reached a peak velocity of 3.9 kph. This represents both the acceleration and deceleration components in a rear-end impact against a stopped vehicle with the brakes applied.[16,37]

Testing was performed with the approval of the Institutional Review Board of the University of Washington. A total of 28 subjects were tested, from whom 26 intact data records were analyzed. The subjects were recruited from hospital employees and included 14 women (age range 22–64 years) and 12 men (age range 28–50 years). Each subject was seated in the sled, shown in Figure 3, and restrained with lap and shoulder belts. A light plastic headband was placed and secured on the subject's head with an elastic strap under the chin. It contained five accelerometers (PCB Piezotronics Inc., Depew, NY), two uniaxial, measuring X (forward-backward) and Z (upward-downward) accelerations at approximately the level of each

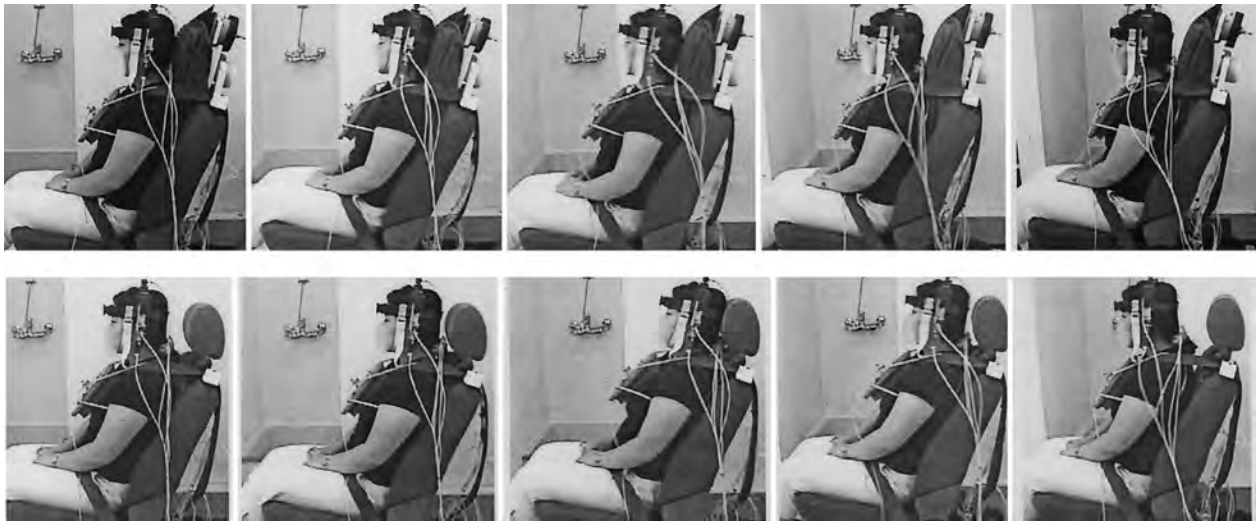

Figure 3. Test subject on sled with headband and chest plate containing accelerometers. Above, sequence showing responses with the modified head restraint. Below, sequence showing the same subject with the standard head restraint in place. Time of images, from left to right: impact, 0 msec, 33 msec, 66 msec, 132 msec, 198 msec.

ear, and one triaxial, located at the apex of the head forming a vertical plane with the accelerometers at both ears. The accelerometers at ear level were located approximately at the center of gravity of the head, in the sagittal plane.[41]

From a previous study,[36] the motions of the head and chest of volunteers in simulated rear-end impacts were observed to occur primarily in the X (forward-backward) and Z (upward-downward) directions or in the sagittal plane, so only X and Z accelerations were measured in this study. Signals were sampled by use of a laptop computer (Powermac G3, Apple Computer Co., Cupertino, CA) with an A/D card and software (Labview, National Instruments, Austin, TX) and were filtered according to SAE J 211, using a fifth-order four-pole Butterworth digital filter implemented in Labview, with cutoff frequencies of 60 Hz and 1 kHz. The instruments on the head were all located on the same rigid body; however, the head translates and rotates in the XZ (sagittal) plane. The resultant at each of the three measurement locations is independent of the orientation of the accelerometers (the local coordinate system) because the resultant can be expressed in an infinite number of coordinate systems. To describe the translation and rotation of the subject's head in the XZ plane, it was transformed into a translation with reference to the lower part of the ear near the temporomandibular joint and an XZ plane rotation about this point. The actual origin for each accelerometer pair was at the intersection of the axes of the X and Z accelerometers. The following transformations were performed: For each of the three measuring locations (right ear, head top, left ear), the resultant was independent of the axis system, and all three axis systems were parallel and located on the same rigid body:

$$\mathbf{Rr} = Xr\,\mathbf{i} + Zr\,\mathbf{k}$$

$$\mathbf{Rl} = Xl\,\mathbf{i} + Zr\,\mathbf{k}$$

$$\mathbf{Rt} = Xt\,\mathbf{i} + Zt\,\mathbf{k} \quad (1)$$

where Rr, Rl, Rt = resultant acceleration vectors at right ear, left ear, and head top, respectively; Xr, Xl, Xt = local X accelerations measured by the accelerometers; Zr, Zl, Zt = local Z accelerations measured by the accelerometers.

For any two points in the XZ plane, such as the right ear and head top, the translational acceleration of the head was defined to be the resultant acceleration for the origin near the right ear, Rr. To determine the rotation about the right ear, the right ear resultant was subtracted from the head top resultant:

$$\mathbf{Rd} = \mathbf{Rt} - \mathbf{Rr} = (Xt - Xr)\mathbf{i} + (Zt - Zr)\mathbf{k} \quad (2)$$

Then the rotational acceleration about the right ear origin is

$$\alpha = Rd/b \quad (3)$$

where α = angular acceleration about the right ear coordinate system; Rd = vector difference in head top and right ear resultants; b = distance from origin of right ear coordinate system and head top coordinate system.

The same calculation was performed using the left ear and head top data. After several calculations were performed, it was observed that head rotation was small, Z accelerations were very small, and the resultants were almost identical with the X translational accelerations. Therefore, the results are described, for ease of understanding, in terms of X translations of the chest and head.

The subject first underwent a rear-end impact sitting upright against the modified head restraint with the air cushion inflated to the subject's comfort level. After the first test, with the occupant still seated, the modified head restraint was removed and replaced with the standard head restraint. The subject's head-to-head restraint horizontal distance was measured. The test was then repeated. Each subject was videotaped to record his or her response. Attempts were made to ensure that the subject was as relaxed as possible during the testing procedure, by discussing the test and by demonstrating the procedure in some cases. Because of the possibility of guarding by tensing neck muscles, especially in repeated testing, the modified head restraint was tested first. The protective effect of tensed muscles, more likely in the second trial, would benefit the results with the standard head restraint.

Peak accelerations and the time when they occurred were derived directly from the test record. The data were then integrated twice to obtain displacement, and the chest-to-head differential displacement at each time point was determined. Statistical analysis was performed using a repeated measures analysis of variance (Stata, release 6, College Station, TX) with the Box conservative correction factor to correct for lack of independence of the subjects. Peak accelerations, time difference between peak accelerations, and the peak head-to-chest displacement difference were analyzed. The time difference between peak accelerations was the measure expected to account for the differential motions between torso and head during impact.

Results

Impact Properties of Seats With a Standard and a Modified Head Restraint

Figure 4 shows a time history of the angular displacement of the pendulum as it impacted the standard restraint, and Figure 5, the modified restraint. There was significantly less rearward angular displacement of the modified head restraint, and less rebound of the pendulum. Table 1 shows that the mean deflection and rebound of the antiwhiplash head restraint were significantly lower (deflection = 4.6 degrees for the prototype *vs.* 14.7 degrees for the standard head restraint, rebound = 12.1 degrees *vs.* 30.6 degrees, n = 6) and the stiffness, 267 N/degrees *vs.* 56.8 N/degrees) significantly greater.

Testing of Human Volunteers With a Prototype Head Restraint Compared With a Standard Head Restraint

The reproducibility of the experimental protocol was tested by subjecting two volunteers to multiple sequential impacts. Table 2 shows the results from one volunteer who received six sequential impacts with the modified head restraint, the overall coefficient of variation for measurements of acceleration and chest-to-head peak acceleration time differences was 0.21. All subjects underwent the two sequential impacts without incident. Subsequent symptoms consisting of some minor neck stiffness later that day or the next were reported by three subjects. All symptoms resolved within a week. In two cases the data were partially lost, so the data presented are taken from 26 trials.

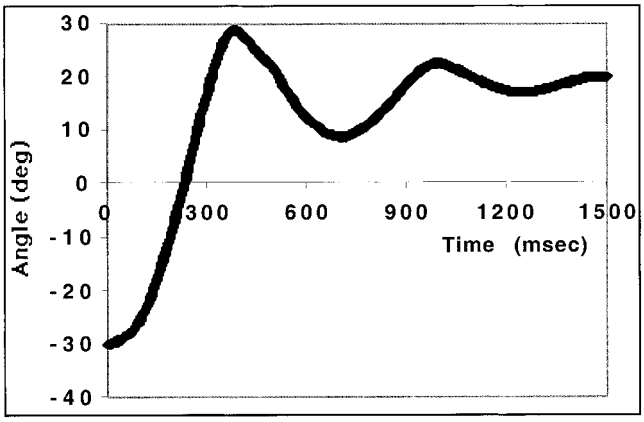

Figure 4. Time history of the angle of the pendulum arm impacting against a standard head restraint with the seat back fixed.

As shown in Figure 3, with the standard head restraint the subject's chest was initially thrust forward while her head was falling backward toward the head restraint. Then her head was thrust forward as the chest motion stopped. With the modified head restraint, both the subject's head and her torso were in contact with the seat, and the initial chest-head differential motion was reduced. Figure 6 shows raw time history data from the acceleration measured at the head top in the X (forward) direction, with the modified head restraint. Figure 7 represents the corresponding tests with the standard head restraint in place. Overall, subjects using the standard head restraint showed a considerably larger variation in responses with peak head accelerations up to 8*g*. By contrast, with the modified head restraint in place, acceleration peaks were considerably smaller, although not statistically significantly different ($P = 0.10$), and the peaks were broader.

Figure 8 demonstrates that the time difference between the occurrence of the peak acceleration of the subject's torso because of contact with the seat, and of the head by the head restraint, was reduced from a mean of 61.3 msec to 22.5 msec between the head top and the chest, from 68.2 msec to 5.9 msec between the right ear and the chest, and from 64.7 msec to 7.8 msec between the left ear and the chest. This time difference in acceleration peaks was demonstrated also in the difference in displacement between the chest in the X direction and the head, shown in Figure 9. The mean chest-to-head displacement with the standard head restraint decreased from 74.6 mm to 40.4 mm at the head top, from 48.0 mm to 20.4 mm at the right ear, and from 48.8 mm to 21.0 mm at the left ear. Examples of the difference in chest-to-head X direction displacement are shown in Figure 10. With the modified head restraint, the chest and head moved fairly uniformly and the initial positive displacement (chest moving forward relative to the head) was small. Later in the test, the chest forward motion stopped while the head moved forward off the head restraint and over the chest (negative displacement). By contrast, with the standard head restraint in place, the chest had greater forward motion compared with the head in the X direction.

Discussion

In this study, it was found that the sagittal plane X direction thrust of the chest before the head, modeled in cadaveric studies, occurred consistently with the occupant sitting in a typical automotive seat with the standard head restraint. The chest-to-head displacement was most likely caused by the thrust acting earlier on the torso, which was initially in contact with the seat back, compared with the thrust on the head, which occurred later because the volunteer's head was initially not in contact with the head restraint. Changing these contact characteristics, specifically having the head and torso both in contact with the seat back initially, decreased the time difference between peak chest-to-head accelerations and also decreased the chest-to-head differential displacement. Subjectively, the participants clearly indicated that the modified head restraint resulted in a more tolerable impact, although interestingly this was not because of differences in the peak accelerations (which were not significantly different) but probably because their torsos and heads moved more uniformly.

Several recent biomechanical studies have proposed different mechanisms of cervical spinal tissue damage. Reports of posterior neck tenderness after rear-end impact are consistent with measurements of muscle electromyographic outputs by Szabo and Welcher[35] that were well beyond maximum voluntary contraction levels in some volunteers subjected to rear-end impacts. This would explain at least some initial symptoms, though not chronic pain. Panjabi et al[24] used a cadaveric model with the lower cervical spine fixed to a sled and a mass representing the head. At higher impacts, the S shape of the cervical spine was demonstrated with the lower vertebra moving forward under the head mass while it remained transiently upright. Starting at 4*g*, the C5–C6 extension range of motion became greater than that considered physiologic, which the authors proposed having the potential to cause soft tissue injury. Raynak[28] mea-

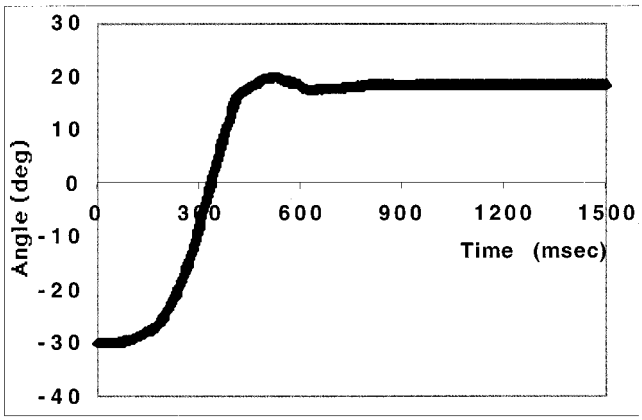

Figure 5. Time history of the angle of the pendulum arm impacting against a modified head restraint with the seat back fixed.

Table 1. Summary of Impact Properties of Standard Automobile Head Restraints and a Modified Head Restraint Applied to the Same Seats

Property	Standard	Modified	*P*
Deflection (degrees)	14.7 (7.3)	4.6 (3.4)	0.02
Rebound (degrees)	30.6 (12.8)	12.2 (8.3)	0.04
Stiffness (N/degrees)	57 (25)	267 (240)	0.02

Error bars indicate 1 standard deviation (n = 6).

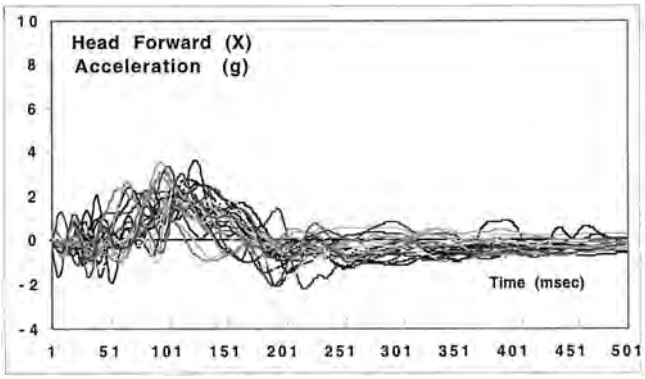

Figure 6. Cumulative time histories from 26 trials for head top forward (X) acceleration (g) with the modified head restraint.

sured transient spinal canal and neural foraminal occlusion in cadaveric specimens. Foraminal occlusions decreased to about 80% of normal intact areas under impact. Eichburger et al[7] consider that increased transient fluid pressure in the spinal canal may result in soft tissue injury and measured transient local pressures from 50 to 200 mm Hg. Nerve root compression would result primarily in radicular pain, which has been found to occur in 84 (20%) of a sample of 432 victims of actual rear-end impacts.[39]

The facet joints have been considered to be a source of symptoms after whiplash. Lord et al[14] showed that direct injection into the facet joints, using a placebo control, caused immediate pain relief in about 60% of a patient population seeking treatment after whiplash injury. Yoganandan et al,[40] using a cadaveric head and neck model, monitored facet surface orientations using markers and high-speed videography. During the transient S phase of the spine they found differential sliding and compression of the facet surfaces of such magnitude that the superior facet angled downward and displaced posteriorly relative to the inferior facet. They hypothesized that this mechanism may cause facet joint pinching. Using volunteers in a sled apparatus that rolled down a ramp into a barrier, Ono et al[23] and Kaneoka et al[13] performed high-speed radiograph analysis on the subjects' cervical spines. They showed that initial abnormal extension of the lower cervical spine occurred and proposed that during whiplash the instantaneous axis of rotation of the C5–C6 segment shifts superiorly, thus altering the facet contact path and causing facet pinching. Alternatively, Siegmund et al[30] applied quasistatic combined posterior shear and compression loads at C3–C4 and C5–C6 and measured capsular strain, using a grid of optical markers on the facet joint capsule. Principal strains during testing reached 33% of catastrophic strains and 61% of subcatastrophic strains measured in separate loading to failure, indicating that loads in the range of volunteer tests could cause mechanical damage to the facet capsule. Deng et al[6] performed high-speed radiograph studies on whole cadavers subjected to rear-end impact. They found a transient S shape to the cervical spine and, from landmarks on the radiographs, measured facet capsular strains up to 60% (at C5–C6 with an upright seat back). The location of the facet capsule strains varied with the angle of inclination of the seat back. They observed that "countermeasures for this injury prevention must deployed very early after impact," which is consistent with the present authors' proposal of a closely conforming head restraint. These studies, while differing in their view of the possible tissues that might be damaged during whiplash, are consistent in the observation that the primary mechanism causing nonphysiologic motion and potentially biomechanical tissue damage is the transient S curvature of the cervical spine.

Table 2. Reproducibility Comparison of Data From 6 Sequential Tests on One Subject, at Three Different Locations on the Head, Comparing Peak Acceleration and Time of Occurrence of This Peak

		Head Top		R Ear		L Ear	
Test	Chest Peak Acceleration (*g*)	Peak Acceleration (*g*)	Head-Chest (msec)	Peak Acceleration (*g*)	Head-Chest (msec)	Peak Acceleration (*g*)	Head-Chest (msec)
1	1.81	2.68	25	1.48	−9	2.30	11
2	1.86	2.74	25	1.82	−12	2.94	9
3	1.90	2.64	19	1.97	−15	3.25	2
4	1.88	2.34	21	1.58	−10	2.67	6
5	1.95	2.02	28	1.54	−11	2.66	6
6	1.90	2.30	19	1.84	−15	2.88	0
Mean	1.88	2.46	22.83	1.70	−12.00	2.78	5.67
SD	0.05	0.28	3.71	0.20	2.53	0.32	4.13
Coefficient of variation	0.02	0.11	0.16	0.11	−0.21	0.12	0.73

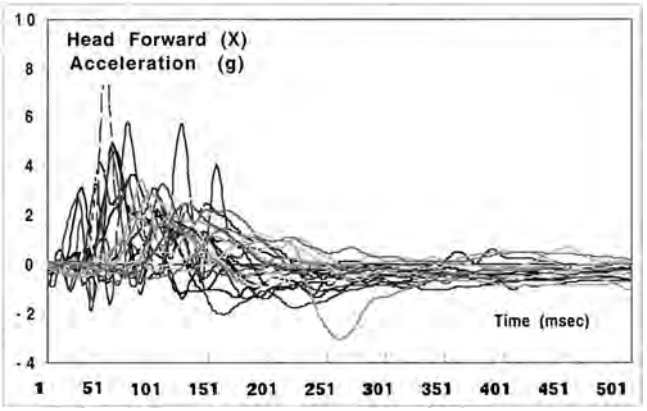

Figure 7. Cumulative time histories from 26 trials for head top forward (X) acceleration (g) with the standard head restraint.

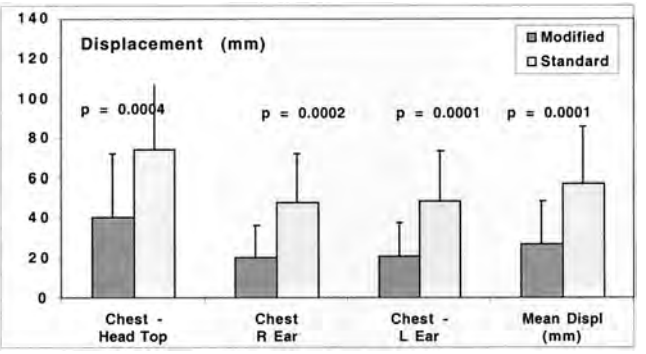

Figure 9. Peak displacement difference in the X direction between chest and head top, left ear, and right ear. Error bars indicate 1 standard deviation; n = 26; p values derived from analysis of variance.

Several other investigators have studied the properties of seats in rear-end impact. Svensson et al[33] found, in testing five different seats using a Hybrid III dummy with a less rigid (RID) neck, that some seats have soft back cushions so that the torso sinks back into the cushion, bringing the head closer to the head restraint, whereas other seats bend backward in response to torso impact so that head-to-head restraint distance initially increases. Subsequently, they designed the whiplash injury protection seat (WHIPS) with a yielding hinge between the seat back and the seat base to absorb some of the impact energy of the seat back–torso impact during the rear-end collision. This seat was found to significantly lower torso accelerations[15] and, in separate testing compared with standard seats, with three human volunteers, to lower both peak accelerations and head-to-thorax displacements.[43] Wiklund and Larsson[44] took a different approach, designing a lever mechanism into the seat. When the torso pressed against the seat back, the lever moved the head restraint forward, closer to the head. Using a Hybrid III dummy with the RID neck, they showed that the head rearward displacement with respect to T1 was reduced from about 35 mm to about 20 mm. Watanabe et al[42] tested a similar system to that described by Wiklund and Larsson[44] along with reducing the stiffness of the upper seat back region. They found that the neck injury criteria, a measure of the relative torso-to-head motion, tended to decrease with decreasing upper seat back stiffness, along with neck rotation angle. In the studies by Welcher et al[43] with three volunteers and Watanabe et al[42] with a single volunteer, head-to-chest linear displacements for human volunteers were reduced to about 20 mm with their seat modifications, which was very consistent with the results of the present study. Neither of the other studies, however, presented sufficient data to establish statistically the differences measured.

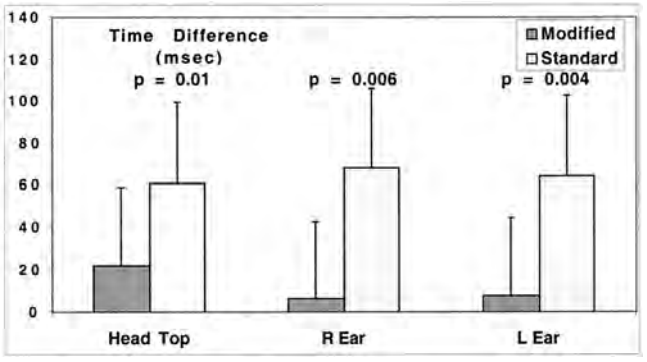

Figure 8. Time difference (milliseconds) between peak X (forward) acceleration acting on the torso from thrust by the seat and on the head from contact with the head restraint with the standard and modified head. Error bars indicate 1 standard deviation; n = 26; *P* values derived from analysis of variance.

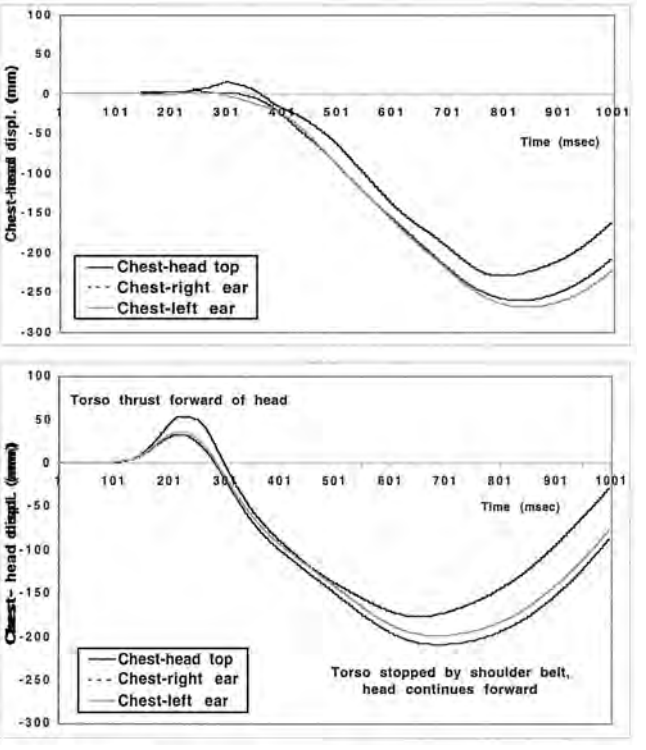

Figure 10. Displacement differences between the chest and head top, left ear, and right ear with positive value indicating the chest moving forward of the head and negative value as the head rebounds forward over the chest (n = 26). Above, modified head restraint. Below, standard head restraint.

This study also was focused not on designing a new seat but on making relatively simple modifications to the seats of vehicles already on the road.

This study produced kinematic results consistent with other studies in the literature. For example, Siegmund et al,[29] in their lower-speed test, generated a vehicle speed change of 4 kph in actual car-to-car collisions with volunteers. The average peak head acceleration reported from 42 volunteers was 3.5*g*, and peak chest acceleration was 2*g*. The chest peak to head peak acceleration time difference was 104 msec. The peak head angular displacement of their volunteers was about 15 degrees. In this study, the speed change of the sled resulting from impact of the pendulum was 3.9 kph. The resultant peak head X acceleration was about 3*g*, and chest acceleration was about 2*g*. The angular displacement of the heads of volunteers in the present study was only about 10 degrees, because even with the standard head restraint, volunteers were sitting fairly close to the head restraint (within about 5–7 cm). The peak chest-to-head acceleration time difference was about 65 msec with the standard head restraint.

As with any study, the limitations of this investigation should be recognized. A rear-end impact experience was recreated as closely as possible within the laboratory setting by the use of an automotive seat with head restraint, and a sled with a bumper to create impacts similar to those of car-to-car collisions, and including the deceleration effect because the occupant, if at a stop when hit, would normally have the brakes applied. Probably the major difference between this study and a real-world impact is that these subjects were obviously aware that an impact would occur, so the element of surprise was missing. Muscle tensing alters impact response.[10,11] An attempt was made to accommodate for this possibility by testing the modified head restraint first, on the supposition that the subject would be more likely to be guarding for the second impact. Thus, muscle tensing, which would reduce head motion, would enhance the data of the second impact with the standard head restraint. In fact, a few subjects' heads never hit the head restraint in the second test, which could indicate some guarding, but the chest thrust and chest-to-head differential motions were still present. A second consideration is that the X acceleration data only approximates the actual resultants. However, the Z accelerations were small enough that the differences between X accelerations and resultants were minor. The authors thought it would be easier to understand the data considering only the primary (X) direction of motion, especially in comparison with cadaver sled tests in which the "torso" (the sled) is constrained to the X direction.

If chronic whiplash is associated with factors other than the acute injury caused by the forces applied to the cervical spine during the impact,[3,9] then this proposed passive intervention may not affect chronic whiplash, even if it reduces the magnitude of the acute injury. The goal of this study was simply to determine whether relative head/torso motions during rear-end impact could be influenced by seat and headrest design. Within the scope of this study, it was not possible to predict the outcome of either acute or chronic whiplash in terms of type or duration of symptoms. It was possible only to describe the changes to kinematics of the spine. The only way to address the outcome of this proposed passive intervention would be to monitor a group of drivers with the intervention and compare their rates of whiplash with those of a control group. However, before this step is taken, the fundamental mechanics of the intervention and the effects of modifications to the seat and head restraint must be assessed. This assessment was the goal of this study.

This study demonstrates that in a rear-end impact with human subjects, there is a measurable time lag between the peak acceleration of the chest, which occurs first because it contacts the seat back, and the head, which occurs later when it impacts the head restraint. This results in a measurable differential displacement, with the chest thrust forward under the head. It is possible to alter this response of an occupant to a rear-end impact by modifying the properties of the head restraint relative to the seat, specifically by creating more uniform contact between the torso and the seat and between the head and the head restraint. If the mechanism of whiplash injury is related to the differential chest-to-head motion, then it may be possible to produce devices with the potential to reduce whiplash injury in these impacts.

■ Key Points

- In several recent studies of the kinematics of the cervical spine of specimens during rear-end impacts, a transient S shape of the spine resulted while the head remained upright, which may result in nonphysiologic intervertebral motions and tissue strains.
- Testing of volunteers was performed to determine how they interacted with the seat and either a standard or a modified head restraint. The modified head restraint was closer to the head, was stiffer, and absorbed impact energy.
- There was a measurable time difference between peak chest and peak head accelerations, which resulted in the chest being thrust forward by the seat back before the head was thrust forward by the head restraint.
- The modified head restraint significantly reduced the contact time difference and therefore decreased the relative chest-to-head forward motion.

Acknowledgment

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Point of View

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The goals of this study are an intuitive response to the prevention of whiplash-associated disorders based on what is known about head/neck kinematics during a rear-end collision. In principle, injury should be reduced if the relative motion between the head and torso is minimized. However, this is easier said than done because it is not known what the reduction should be or how quickly this should be accomplished during the impact, because it is not known for sure where the pain is coming from. What this article shows is that it is feasible to reduce this relative motion, but it is not known whether this reduction alone affords sufficient benefit to the victim. Recent data show that even with the head right up against the headrest, the cervical spine can still change its curvature.

This point of view addresses wider issues than those discussed in this article. However, it is one of many recent articles that were written in an attempt to solve the problem of whiplash-associated disorders. The important issue of pinpointing the source of pain has not been fully addressed. Many hypotheses have been proposed, and even though a consensus seems to be building to point the finger at the cervical facet joint capsule, no basic research into the neurophysiologic basis of facet pain appears to have been done or even begun. The clinical study by Lord et al[1] should provide the needed momentum to get the job done.

Another issue is related to various forms of rear-end impact dummies, which have a detailed representation of the cervical spine, and to the many devices being proposed to prevent whiplash-associated disorders, such as the one proposed in this article. Because of their inability to identify the source or sources of pain, researchers are putting the cart before the horse and are trying to find a cure without knowing the cause. Such an approach not only is inefficient and costly but also carries with it the risk of not benefiting the crash victim. It behooves the biomedical engineer to heed the clinical maxim: "Above all, do no harm."

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Response to Point of View

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We would like to thank you and the reviewers for accepting our manuscript, "The Response of Human Volunteers to Rear-End Impacts," for publication in *Spine*. I have read the point of view written by Dr. King to accompany our article and would like to respond to it. Dr. King makes several valid points in his thoughtful comments on our article and on the more general approaches being taken in the field to address the problem of whiplash.

We and others in the field of biomechanics (including Deng et al[1]) have made the basic assumption in our studies that whiplash symptoms are at least initiated by a mechanical event, the rear impact, and develop from the resulting accelerations and forces exerted on the cervical spine and their effects on soft tissues. If this is the case, then it appears reasonable to address a solution to this problem by focusing on how to reduce spinal forces and displacements, regardless of the specific source of pain. It also seems reasonable to us that reducing cervical spine forces and displacements lies within the "do no harm" philosophy, with which we certainly agree. The basic point of the article was that during a rear impact, spinal forces and displacements are affected by the manner in which contact occurs between the occupant's body and the seat and head restraint, and that this can be altered. There was no specific indication that this would prevent whiplash.

Determining the root cause of neurophysiologic pain in whiplash is, of course, important. It is hard to imagine, though, an animal model that will satisfy the requirement that it respond in a similar manner to an upright seated human under rear impact, including appropriate interactions with the seat and head restraint, and also provide measurable data on the resulting pain response. Although that model is yet to be developed, biomechanical studies in the literature support the concept that the geometry of the head restraint can be improved. These concepts will be part of modified federal regulations designed to provide increased protection against whiplash by increasing the height of the head restraint and its proximity to the back of the occupant's head.[2]

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Parametric Analysis of Vehicle Design Influence on the Four Phases of Whiplash Motion

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Objective. *The objective is to establish a basis for motor vehicle test requirements that measure component contributions to Whiplash Associated Disorders (WAD).*

Methods. *Selected vehicle design features are evaluated with regard to their relative contributions to WAD measures. The motion of the occupant cervical spine associated with WAD is divided into four phases: retraction, extension, rebound, and protraction. Injury measures from the literature (NIC, extension moment, N_{km}, and flexion moment) represent the injury potential during each of these phases. Four vehicle design factors that affect WAD motion (vehicle stiffness, seat stiffness, head restraint height and head restraint backset) were evaluated for their contributions to the injury measures. A detailed 50th percentile male model with a biofidelic neck was used in a 100-run Monte Carlo analysis of a rear impact, varying the design factors across the values documented in the literature. Total energy was held constant and Delta V was 10 kph.*

Results. *Vehicle stiffness has a strong influence on the retraction (70%), rebound (43%), and protraction (47%) phases. Headrest backset demonstrates a strong influence on the extension (49%) and rebound (39%) phases.*

Conclusions. *For WAD protection rating, the vehicle should be viewed as a system whereby the complex interactions among the vehicle, seat, and occupant characteristics all contribute to the WAD potential.*

Keywords Cervical Spine; Whiplash; Mathematical Models; Multi-Body Dynamics; Sensitivity Analysis

Whiplash Associated Disorders (WAD) are the most frequent injuries resulting from rear-end motor vehicle accidents, so it is desirable to quantify how vehicle features can be designed to reduce them. Establishing how vehicle design parameters affect WAD will lead to testing vehicle components for WAD injury potential and will, in turn, influence future vehicle design decisions through publicly-disclosed ratings similar to those provided for high-impact crash protection (Langwieder, 2000). The design and rating of vehicles regarding whiplash injury protection may prove challenging because multiple design factors are likely to affect this motion. Head restraint position (Davidsson et al., 1998; Zuby et al., 1999; Siegmund et al., 2001), head restraint stiffness (Chhor et al., 1996; Eichberger et al., 1996; Hofinger et al., 1999), seat energy-absorption characteristics (Svensson et al., 1993; Nilson et al., 1995; Haland et al., 1996; Hofinger et al., 1999; Muser et al., 2000; Welcher & Szabo, 2001; Viano, 2003) and vehicle structural characteristics (Robbins et al., 1974, Nilson et al., 1994; Zuby et al., 1999; Linder et al., 2003) are all identified as contributors to WAD.

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Some studies have evaluated the discrete effects of vehicle design factors on one or more injury criteria. For example, studies have been performed that evaluate the influence of multiple vehicle design factors on NIC (Eriksson & Bostrom, 1999) or the influence of a particular seat design on multiple injury criteria such as NIC and N_{km} (Muser, 2000). However, no studies have quantified the varying effects of primary vehicle design factors across all WAD phases. Given the current initiatives to develop rear impact test standards to assess vehicle WAD protection (Langwieder, 2000; Tencer et al., 2004), it is important to understand the injury potential for each vehicle design factor on each phase of WAD motion. The current study was performed to better understand the complex relationships between the vehicle design factors known to affect WAD and the different phases of WAD motion. The results are helpful in determining which vehicle design factors should be evaluated in the testing and in establishing criteria that should be used to assess the test results.

OCCUPANT MOTION IN REAR IMPACTS

The occupant motion occurring during rear impacts has been divided into four phases: retraction, extension, rebound and

protraction. Because each phase exhibits different occupant responses or motions, the injury-causing stresses are likely associated with different mechanisms. The whiplash injury measures associated with the different occupant responses have received varying definitions from the biomechanics community (NHTSA, 2000; Welcher & Szabo, 2001; Viano & Davidsson, 2002). For this study we have selectively adopted phase injury measures from among those that have been proposed by other researchers.

Retraction Phase

In the retraction phase, the upper torso is pushed forward by the seat back while the occupant's head remains nearly stationary. Consequently, the cervical spine has been shown to take on an S-shape by forcing the upper cervical spine into flexion and the lower cervical spine into extension (Eichberger et al., 1996; Ono et al., 1997; Ono et al., 1998; Svensson 1998; Yoganandan et al., 1998). The retraction phase ends when the maximum relative translation of the head and torso is reached. An important injury mechanism proposed to be associated with this motion is a pressure spike in the spinal canal caused by the rapid relative translation of C1 with respect to T1 during retraction (Aldman, 1986; Svensson et al., 1993; Bostrom et al., 1996). The Neck Injury Criterion (NIC) was explicitly developed to quantify the risk for WAD based on this mechanism (Bostrom et al., 1996). NIC values greater than 15 m^2/s^2 are believed to be associated with the potential for long-term symptoms (O'Neill et al., 1972; Bostrom et al., 1996; Zuby et al., 1999), although some believe this may be high (Croft et al., 2002). A NIC reading of 12 m^2/s^2 appears to be about as high as researchers are willing to expose human subjects (Hell et al., 1999; O'Neill, 2000; Welcher & Szabo, 2001), although a few values up to 19.8 m^2/s^2 are documented in the literature (Welcher & Szabo, 2001). NIC is chosen in this study to quantify the influence of design parameters on injury outcome in the retraction phase, even though there is some disagreement (Ono et al., 1997, 1998) that the predominant mechanism is the pressure spike NIC mathematically represents. The methodology developed in this paper can be extended to investigate the influence of design parameters on other injury measures (such as those used by Ono) or combinations of measures.

Extension Phase

The extension phase begins after the head reaches maximum translation and then rotates rearward. This rotation causes the upper cervical motion segments to join the lower motion segments in extension orientations. An important injury mechanism associated with this motion is hyperextension, first cited for WAD causation as early as the 1950s (Severy et al., 1955; Severy, 1960, 1970; MacNab, 1964, 1965). Injury potential is measured by the extension moment exerted at the atlanto-occipital joint or by exceeding the normal range of motion. The former measure is used herein. Extension moments exceeding 30.5 Newton-meters (Nm) are believed to present significant potential for injury, based primarily on the work of Mertz and Patrick (1967, 1971). The Society of Automotive Engineers (SAE) Standard J885 incorporates this limit into its guidelines for human subject testing (SAE, 1986). Only one set of volunteer tests that approached or exceeded this limit was found in the literature (Mertz & Patrick, 1967).

Rebound Phase

The rebound phase occurs after the occupant's head contacts the head restraint. Head restraint rebound causes the highest head translational accelerations to occur, as well as peak axial and shear forces. The cervical spine is known to be particularly vulnerable to injury when significant torque moments are combined with significant shear or axial forces. The N_{ij} criterion is currently used by the National Highway Transportation Safety Administration (NHTSA) to assess neck injury potential in higher impact crashes by combining neck axial forces and torques (Kleinberger et al., 1998). In a similar manner, the N_{km} criterion has been proposed to evaluate the effect of combined shear forces and moments (Muser et al., 2000) for assessing the potential for WAD injury. We have elected to use N_{km} as the rebound phase measure.

Protraction Phase

The protraction phase occurs after rebound when differential motion between the head and torso are reversed. This phase becomes significant when the forward motion of the upper torso is arrested by the seat belt shoulder strap. Mechanisms associated with this motion are similar to those in the retraction and extension phases when the cervical spine transitions from an S-shape toward full flexion. A pressure spike in the spinal canal, which is reflected in the NIC, has been proposed as a potential mechanism for whiplash injuries occurring in frontal impacts (Bostrom et al., 2000). Moments, axial forces, and shear forces are also caused by forward motion of the head relative to an upper torso restrained by a shoulder harness. Difficulties in deriving these values from rear impact ATDs have led many researchers to look at head and T1 rebound velocities to assess rebound and protraction-phase effects (Muser et al., 2000). We selected the flexion moment for this study.

INFLUENCE OF DESIGN FACTORS ON WAD

The influences of key vehicle design features on WAD measures, are investigated by performing a parametric analysis using a detailed mathematical human model and a vehicle model that allows for the effects of vehicle structural factors and seat and headrest factors. Ultimately, we will establish boundaries for vehicle design parameters that minimize WAD.

The force-deflection characteristics of the two colliding vehicles produce the rear-end impact crash pulse that helps generate the occupant motion. The force-deflection characteristics for low speed rear impacts are determined by the bumper systems of the colliding vehicles and they can vary widely depending on the vehicle design. For this study, various combinations of vehicle force-deflection curves, derived from the literature (Evans, 1998; Gilliard, 1998) or from crash testing (NHTSA,

2000; Sendur et al., 2003), were used to determine expected real-world durations for 10 kph velocity changes.

Another force acting on the occupant comes from the seat characteristics. We selected factors representing seat stiffness, which helps determine the rise time and rebound on the occupant translation and rotational motion, and the headrest, which has significant influence on the extension and rebound phases by affecting the trajectory of the head and neck. All head restraint positioning is based on the positioning system found in the Research Council for Automotive Repair (RCAR) standard, also used by the Insurance Institute for Highway Safety (RCAR, 2001). Several studies have been conducted to characterize the rotational stiffness of different seat designs (Viano & Gargan, 1996; Strother & James, 1998; Molino 1998; Viano, 2003).

METHODS

Hypothesis and Test Setup

A combined mathematical vehicle and occupant model are used to examine WAD injury measures for a well-aligned rear-impact crash. The mathematical modeling employs a representation of the vehicle bumper system, the occupant, and the seat. A 100-run Monte Carlo analysis was conducted, varying key vehicle design factors across their expected range of real-world values. Total energy was held constant and all runs were carried out at a Delta V of 10 kph. The influence of the design factors was assessed using the injury criteria proposed for each phase of the WAD motion.

Description of the Modeling Environment

Multi-body models were constructed in MSC.ADAMS (Sendur, 2002; Sendur et al., 2003). The occupant model represents a 50th percentile male, with individual cervical and lumbar vertebrae. The model has 24 segments and 23 joints (head, cervical spine, torso, lumbar spine, pelvis, upper and lower arms, upper and lower legs, and feet). Occupant component motions are based on appropriate component mass properties, dimensions, joint stiffness and damping friction, and limits obtained from various published sources. The joints are tri-axis hinge joints except for the shoulders and hips, which are spherical. The occupant model is shown in Figure 1.

The human cervical spine model is based on the De Jager model (De Jager, 1996), and is similar to that used on other research studies (Ommaya & Hirsch, 1971; Kroonenberg et al., 1998). The upper spine model includes seven vertebrae (C1–C7), the head and the first thoracic vertebra (T1). The rigid bodies in the upper spine model are connected through two-axis nonlinear viscoelastic joints, which represent the mechanical behavior of the intervertebral discs, ligaments, facet joints, and muscles. The neck joints are permitted movement in the sagittal and lateral planes but not about the spinal axis. The head is permitted motion about all three axes. The force-deflection relationships used in the model are nonlinear, and the moments are non-symmetric for motions about any axis.

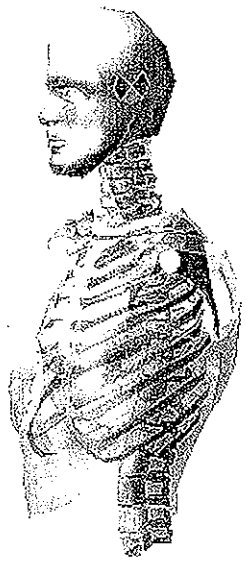

Figure 1 Occupant model.

A similar modeling strategy is taken for modeling the lumbar spine. The lumbar spine is comprised of five vertebrae, L1 through L5. These rigid bodies are connected by three-axis viscoelastic elements, which represent the mechanical characteristics of the inter-vertebral discs, ligaments and muscles. The kinetic characteristics of the lumbar spine were calibrated to the human test data available in the literature by iterating the model until the best match was obtained (Air Force Research Laboratory, 1984). The thoracic spine is modeled as a single segment given the restriction in movement provided by connections to the rib cage.

The seat model is comparable to other models published in the literature (Eriksson & Bostrom, 1999; Welcher & Szabo, 2001). The seat model is comprised of three bodies—a seat bottom, seat back, and a head restraint. The seat back is oriented with respect to the seat bottom at a specified angle, and is constrained with a hinge joint. The seat characteristics represented include the seat back hinge stiffness, the stiffness of the seat cushion and the friction coefficients between the occupant body parts and seat (including pelvis-seat bottom, pelvis-seat back, lumbar spine-seat back, torso-seat back). A friction coefficient of 0.7 is used for contact between the occupant and the seat, except for head contact with the head restraint, which is modeled using a friction coefficient of 0.5 (Kleinberger et al., 1999). The head restraint is modeled as a deformable body fixed to the seat back with a specified backset and headrest height (Kleinberger et al., 1999).

To establish a level of confidence in the kinematics of the occupant model, the kinematic responses of the occupant model to crash motions are compared with the responses of human volunteers subjected to sled accelerations simulating rear impacts at a Delta V of 9 km/h (Cappon et al., 2001). The comparison dataset consists of 43 tests using 19 human subjects, and Hybrid III and RID2 ATDs. The seat back is oriented at 25 degrees backward inclination and a regular 3-point belt is used. The performance

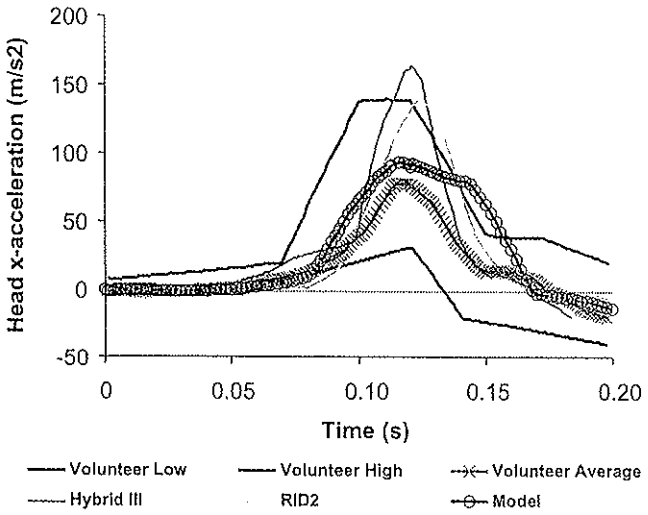

Figure 2 Validation-head x acceleration.

of the model on head translational acceleration and T1 rotation angle is compared with the average human test subject response in Figures 2 and 3, respectively. The lower and upper corridors from live human tests are plotted, along with the average human response. Hybrid III and RID2 responses are shown on the plots to show that they are not biofidelic. The model response fits reasonably well within the lower and upper corridors, especially the peak points of these outputs, which are considered important to injury potential (Mertz & Patrick, 1971; Ommaya & Hirsch, 1971; O'Neill et al., 1972; Goldsmith & Ommaya, 1984; Bostrom et al., 1996). In addition, the shape of the human model response is similar to the live occupant response of the volunteer group. This shows the model's bio-fidelity to live human subject responses, some of which the Hybrid III and RID2 ATDs fail to capture. Differences between the model and data can be attributed to the uncertainty in the parameters used in the live tests, particularly the head restraint position, and variations in the neck strength and muscle response times of the various volunteers (Szabo & Welcher, 1996; Brault et al., 1998; Magnusson et al., 1998).

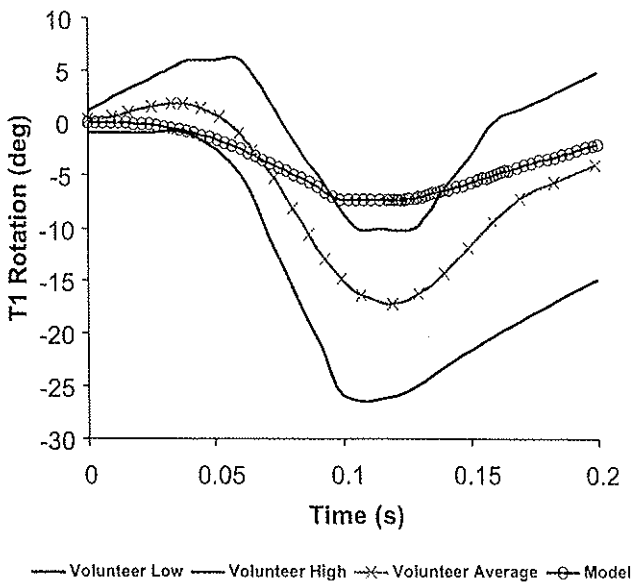

Figure 3 Validation-T1 Rotation Angle.

Variation in Vehicle Structural Factors

Real-world crash pulses are complex shapes that do not lend themselves well to understanding sensitivity at a high level. So, idealized pulses were created using the durations representative of the more complex force-deflection curves, which range from 30 ms to 180 ms with peak accelerations ranging from 2.5 g's to 14.9 g's (see Figure 4). Since total energy is being held constant, the crash pulse effect is reduced to a single variable, duration. A normal distribution cutoff at the first standard deviation is used to approximate the real-world distribution of impact durations. Although the pulse shapes used in this study are idealized, they represent the minimum complexity of the relationships between the vehicle design factors and WAD injury mechaniss.

Variation in Seat Factors

The headrest backset is varied from 3.1 cm to 14.7 cm, using a normal distribution based on an observational study (Cullen et al., 1996). Head restraint height is varied from 3.8 cm to 13 cm using a normal distribution also based on the same study. Because of the trend toward stiffer seats over the last several years, the values found in Viano and Gargan's work (1996) are used for seat stiffness variation, using a normal distribution ranging from 35 Nm/deg to 85 Nm/de.

Procedure

Four predictor variables related to automobile design parameters were selected based on published results and engineering judgment: crash duration, headrest backset, headrest height, and seat stiffness. Then a systematic search was undertaken to identify the variables that were the best predictors of the individual WAD effects for each WAD phase. Scatter plots were made for each injury measure and each predictor variable to see the most likely relationships and how well the single-variable relationships behaved. All head accelerations are measured at the

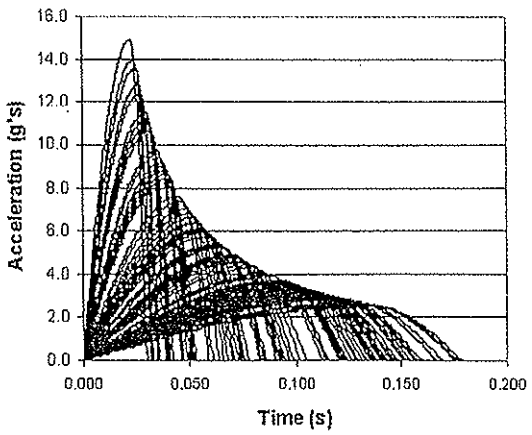

Figure 4 Idealized crashpulses used in Monte Carlo runs.

Table I Injury measure variations from Monte Carlo simulations

Injury measure	Low	High	Average
NIC (m^2/s^2)	1.53	21.22	12.17
N_{km}	0.11	0.58	0.37
Flexion moment (Nm)	2.52	27.44	11.08
Extension moment (Nm)	0.50	11.8	7.68
Tension (N)	1	500	151
Compression (N)	39	372	149

center of gravity, while forces (shear and axial) and moments (extension and flexion) are measured at the Occipital Condyles (OC). The OC is also used as the reference for the top of the cervical spine to derive extension and flexion moments. Care was taken to insure that all injury measures occurred in their proper phases. Trial single-variable regressions were performed. When more than one predictor variable showed good correlation or a high regression coefficient, multiple regression techniques were applied. The end result is a set of numbers quantifying the contribution of each predictor variable during each WAD phase.

RESULTS

Most of the runs result in contact between the head and head restraint. Head accelerations range from 4.3 g's to 14.2 g's. Head angular accelerations range from 73 rad/s^2 to 1955 rad/s^2, with a few runs exceeding the 1800 rad/s^2 often used as a threshold for non-contact concussion (Ommaya & Hirsch, 1971). NIC ranges from a low of 1.53 m^2/s^2 to a high of 21.22 m^2/s^2 with over a third of the results exceeding the proposed threshold of 15 m^2/s^2 (Muser et al., 2000). NIC frequency is shown in Figure 5.

Tension forces vary from 1 Newton (N) to 500 N with 13% of the runs exceeding the limits for human subject testing specified by SAE J885. Compressive forces vary from 39 N to 372 N with 9% of the runs exceeding the SAE J885 recommendation (SAE, 1986). Neck moments also vary from a low of 2.5 Newton-meters (Nm) to a high of 27.4 Nm; none exceed the limits for human subject testing specified by SAE J885. Low, high, and average measures are summarized in Table I.

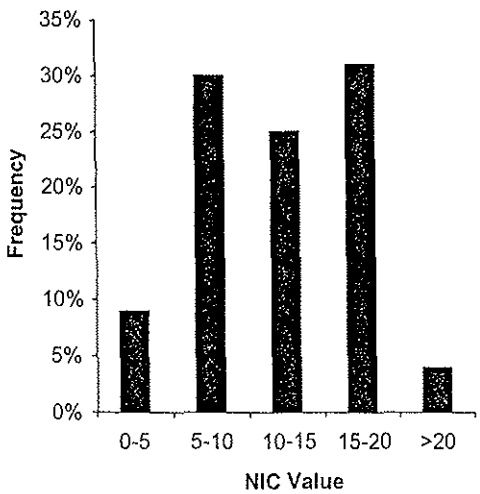

Figure 5 NIC frequency plot.

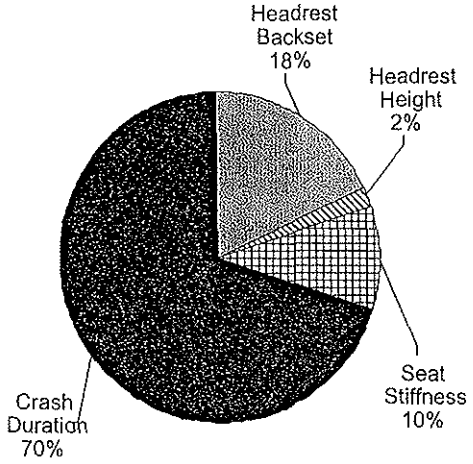

Figure 6 Sensitivity-retraction phase.

Retraction Phase Sensitivity

In the retraction phase, NIC is found to be most sensitive to crash duration, which in this study defines the severity of the crash pulse in terms of duration and peak acceleration. NIC also shows significant sensitivity to head restraint backset. Seat stiffness and head restraint height have little influence on NIC. The influences of all four design factors on the retraction phase, as represented by the NIC criterion, are shown in Figure 6.

Extension Phase Sensitivity

Neck extension moment is used to assess the injury potential for this phase. Head restraint backset has the greatest influence on neck extension moment. Extension displays some sensitivity to crash duration, but very little to head restraint height or seat stiffness. The influence of all four design factors on the extension phase, as represented by extension moment, is shown in Figure 7.

Rebound Phase Sensitivity

In the rebound phase, crash duration shows the most influence on N_{km}, followed closely by head restraint backset. Head restraint height and seat stiffness have minimal influence on N_{km},

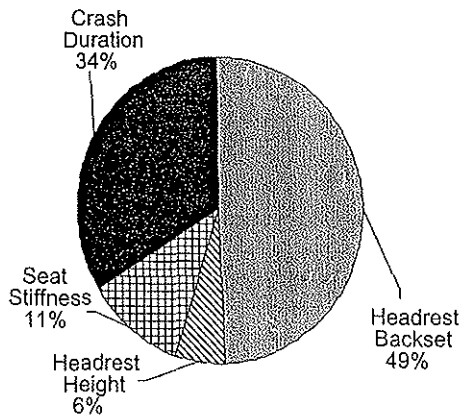

Figure 7 Sensitivity-extension phase.

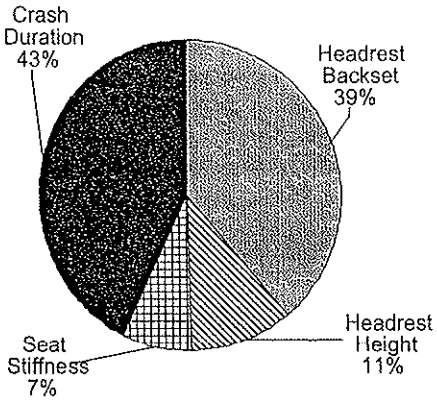

Figure 8 Sensitivity-rebound phase.

although seat stiffness has a significant influence on one of the four measures making up the N_{km} criterion. For N_{km} Flexion Posterior, seat stiffness has an influence of 18%, an influence that is lost when combined into a maximum N_{km} value. Seat stiffness also has an influence of 17% on the shear force posterior measure. The influences of all four design factors on the rebound phase, as represented by N_{km}, are shown in Figure 8.

Protraction Phase Sensitivity

In the protraction phase, measured by flexion moment, crash duration emerges as the most significant influence on injury potential. Head restraint backset has an influence of 26%, while seat stiffness and head restraint height have an influence of 15% and 12%, respectively. The influences of all four design factors on the protraction phase, as represented by flexion moment, are shown in Figure 9.

ANALYSIS AND DISCUSSION

These results first suggest that vehicle design plays an important role in protecting the occupant from whiplash injury and highlights the importance of examining the effects of all key design features on the different phases of the whiplash motion. Different design factors have different levels of influence on the phases of the whiplash motion, as depicted in Figure 10.

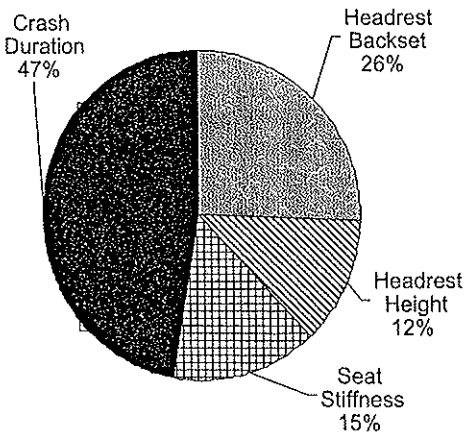

Figure 9 Sensitivity-protraction phase.

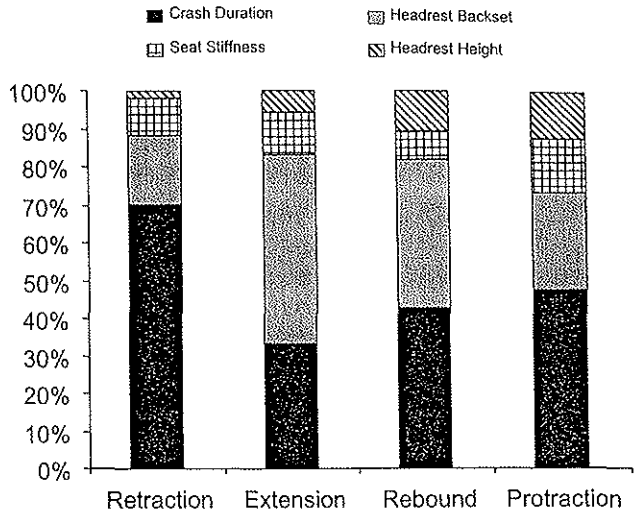

Figure 10 Design sensitivity by whiplash phase.

Second, the results suggest that, in designing and rating vehicles for whiplash protection, it is important to look at the vehicle as a system and not just a single design variable such as seat stiffness or head restraint geometry.

Third, the results of this study also suggest that the multiple phases of whiplash make vehicle design quite challenging. Even with this simplified model, the relationship between design features and proposed injury measures is shown to be complex. It is likely that examination of other parameters known to influence occupant motion would add to the complexity demonstrated by this study. Other factors that were not varied could also be expected to influence injury measures, including seat geometry and friction between the occupant and the seat. The results also highlight the influence of structural force-deflection characteristics in mitigating whiplash. The significant role that vehicle stiffness plays, particularly in the retraction phase, is something that should be considered by testing agencies that rate vehicle performance.

The results agree well with the study by Kleinberger, Sun, Eppinger, Kuppa, and Saul (1999), which demonstrated that reducing the headrest backset position reduces neck injury in rear automotive impacts. Furthermore, this publication demonstrated that decreasing the backset position also reduces the value of the relative displacements, loads (tensile and shear forces) and moments (extension and flexion moments) on the cervical spine. It indicates that backsets of less than 2 inches contribute little to the injury measures: relative rotations, shear and tensile loads, bending moments, N_{km}, and NIC. The Insurance Institute for Highway Safety (1999) reported that little risk of WAD injury exists if both the distance from the top of the head down to the top of the head restraint is less than 2.4 inches and the backset distance is less than 2.8 inches. A study by Volvo reported that when vehicle occupants have their heads against the headrest during impact, no injury occurs (Jakobsson, 1994).

It is likely that, at different crash magnitudes, the influence of vehicle design parameters would change significantly. For example, we show that seat stiffness has little influence on the

injury measures for any phase of motion, although it does have a significant influence on one of the four components of N_{km} (see also Szabo et al., 2003). The influence of seat properties at higher impact magnitudes is well established (Viano, 2003) and it is likely that the relatively low magnitude of this study was simply not enough to significantly invoke the seat stiffness.

This study was based on a well-aligned rear impact with no underride, override, angle, or offset. Other factors known to influence occupant motion were not varied; they include seat angle (Hofinger et al., 1999), occupant/seat friction (Nilson, 1994), seat cushion stiffness (Hofinger et al., 1999; Tencer et al., 2002), head restraint stiffness (Hofinger et al., 1999; Tencer et al., 2002) occupant characteristics (Sieveka et al., 1995; Kroonenberg et al., 1998; DeRosia et al., 2002), occupant position (Nilson, 1994; Matsushita et al., 1994; McConnell et al., 1995), and occupant reaction (Szabo & Welcher, 1996; Brault et al., 1998; Magnusson et al., 1998). It is likely that consideration of these additional factors would have resulted in greater variations among the injury measures. This study was also limited to an impact magnitude of 10 kph. It is hypothesized that the influences of the design factors on the different phases of occupant motion will vary significantly based on impact magnitude. Comparison of the influence of key design factors on the various phases of whiplash at varying impact magnitudes is reserved for a future work. Additionally, this study did not sort out the effects that are purely within the control of the vehicle manufacturer from those that are in control of the occupant; that is also reserved for a future study.

CONCLUSIONS

Using mathematical simulation to examine the relationships between WAD phase-oriented injury measures and selected vehicle parameters, the relative contributions to injury show significant variation among the phases. The results will help obtain optimum vehicle designs that minimize WAD under practical design constraints such as cost. Designers will have to decide how to weigh investment in seat design and features versus bumper system stiffness. The relationships here are given for a single impact speed and crash energy input, and the results may differ when a range of impact energies is considered. The results depend on the validity of the injury measures for each phase. However, the methodology developed in this article can be applied to different injury measures or to combinations of those measures.

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BioRID II manikin and human seating position in relation to car head restraint

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BioRID II manikin and human seating position in relation to car head restraint

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The objective of the study was to compare stature, weight and backset (the horizontal distance (x) between the back of the occupant's head and the front of the seam on top of the head restraint) of the Biofidelic Rear Impact Dummy (BioRID II) to the same variables on seated volunteers in a car. The following methods were used. Data were collected from 154 randomly selected Swedish individuals (78 males and 76 females). The volunteers and the BioRID II were examined in a Volvo V70 car, year model 2003, in three positions: driver (hands on steering wheel), front passenger (hands in lap) and rear passenger. The study results were as follows: the BioRID II was found to correspond approximately to a 35th–45th percentile male in stature (−2 cm), a 35th percentile male in weight (−7 kg), a 96th percentile female in stature (+11 cm) and a 69th percentile female in weight (+8 kg). The BioRID II was designed to represent a male driver. The BioRID II backset corresponded well with the average of the male drivers of its stature. Larger deviations in backset were found for other volunteer sizes and other seating positions. The average backsets were 26 mm for females and 63 mm for males in the front seat positions. The volunteers had larger backset in the driver position (60 mm) than in the front passenger position (29 mm). Smaller differences in backset were seen between the BioRID II and the volunteers in the rear passenger position. This study provides data regarding the occupant size coverage of BioRID II, and unique data regarding backset, of different occupant positions in the car; driver with hands on steering wheel, and front and rear passengers with hands in lap, for female and male in relation to the BioRID II dummy.

Keywords: BioRID II; stature; weight; backset; gender; position

Introduction

Although the pathological cause of whiplash injury is still unknown, there is a large body of knowledge regarding injury-generating factors. Factors causing increased risk include female gender, large backset and driving position. As knowledge about injury criteria increases, the need arises for tools that can be used by car manufacturers to evaluate their designs. Many evaluations can be made using computer simulations, but there will always be a need for real test crashes and occupant substitutes, like the BioRID II dummy. In order to evaluate seat designs in a standardised way, several test protocols have been developed by, for example, the International Organization for Standardization (ISO) [7], the Research Council for Automobile Repairs (RCAR) [13] and the Research Council for Automobile Repairs – International Insurance Whiplash Prevention Group RCAR-IIWPG [14]. These protocols include a procedure for positioning the BioRID II in a seat when performing a dynamic test of a rear impact.

The size and shape of the BioRID II dummy were derived by Schneider [16] using anthropometric data from the University of Michigan Transportation Research Institute (UMTRI). The choice to use the size of an existing 50th percentile male dummy was made in order to simplify the development, as it allowed the use of some existing dummy parts, such as extremities and head [3,4]. Another reason for this choice of size was that it could represent not only a normal-sized male, but also because this size was quite representative of a tall female, being one of the high-risk occupants [10].

New data from a Swedish population were recently presented by Jonsson et al. [9], who studied female and male seat adjustment precision and repeatability. There was a significant difference between the settings for males and females, mainly due to stature differences.

The aim of this study was to analyse how well the BioRID II, when positioned according to the ISO [7], RCAR [13] and RCAR-IIWPG [14] protocols, corresponds in size and backset to data from a randomly selected population of Swedish female and male volunteers, sitting as drivers (hands on the steering wheel) and as front and rear seat passengers (hands in the lap).

Method

Human volunteers

Test volunteers were age and gender stratified, but otherwise randomly recruited from among (i) visitors to the

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Swedish Vehicle Inspection Company (Svensk Bilprovning AB) in Örnsköldsvik, and (ii) citizens in the community of Örnsköldsvik. The Swedish Vehicle Inspection is a body which conducts annual vehicle inspections on the Swedish car fleet of 4.2 million cars; 3.4 million of these cars have private owners SIKA [17]. The inclusion criteria were a valid class *B**** driving license, and pain-free neck, thoracic and shoulder regions. In total, 154 volunteers (78 men and 76 women) participated in the study and were the same individuals as in study [8,9]. Data regarding stature, weight, seat adjustment and backset were collected at the same occasion. The mean age of the men was 46 years (standard deviation SD = 11), and the mean age of the women was 48 years (SD = 11). The volunteers were given written and oral information about the study's aim and procedure before they gave their consent to participation.

Measuring equipment

The test car was a Volvo V70 model, year 2003, manufactured for the US market; both front seats were equipped with an electric power seat, model P2. The volunteers were instructed to adjust the front seat in their preferred way, using the same routine as in an earlier study by Jonsson et al. [8]. Self-selected posture backset was measured with the same equipment and method as described below for the BioRID II.

The manikin used in this study was a BioRID II, version H. A measurement device was constructed to measure head position in the sagittal plane, as compared to a 0-reference point at the head restraint; the same device was used for both manikin and volunteers. The device was constructed from two movable metal rods with a lockable nut at the grade scale in between; see Figures 1 and 2. A second and lower grade scale was mounted on a metal frame which was inserted into the normal position for the head restraint attachments. The position of the movement centre of the lower grade scale was carefully measured in relation to the upper and frontal fabric rim of the head restraint 0-reference point (**A**); see Figure 3. The X- and Y-values were calculated on a laptop on the basis of the known length of the rods, seat back angle (γ) and the values of the two grade scales (α, β) with the following formulae:

Horizontal distance (X) in mm = $(300 \cdot \cos(180 - \alpha) + 400 \cdot \cos(\alpha + \beta - 180)) \cdot \cos(\gamma) + (300 \cdot \sin(180 - \alpha) - 400 \cdot \sin(\alpha + \beta - 180)) \cdot \sin(\gamma) - 248$.

Vertical distance (Y) in mm = $(300 \cdot \sin(180 - \alpha) - 400 \cdot \sin(\alpha + \beta - 180)) \cdot \cos(\gamma) - (300 \cdot \cos(180 - \alpha) + 400 \cdot \cos(\alpha + \beta - 180)) \cdot \sin(\gamma) - 252$

where α is the angle lower grade scale, β is the angle upper grade scale and γ is the angle seat back.

The maximum intrapersonal measuring inaccuracy in reading the grade scales was $\pm 0.5^{\circ}$, leading to a possible measuring error of ± 2 mm in the horizontal direction (X-values) and ± 1 mm in the vertical direction (Y-values).

Measuring points on the BioRID II and seat/head restraint

The measuring point on the BioRID II was the most rearward point of the dummy head, corresponding to the external occipital protuberance on the back of the head on volunteers; see Figure 1. The 0-reference point (A) on the seat was set to the frontal aspect of the seam of the fabric on top of the head restraint; see Figure 3. After BioRID II setup according to the protocols [7,13,14], the head restraints in the front and rear seats were removed, in order to allow *comparative* evaluation of volunteer seating preferences without sagittal guidance from the head restraint.

Test positions and procedure – BioRID II measurements

The BioRID II was measured in (i) driver position and (ii) front passenger position according to the procedure described in the ISO [7], RCAR [13] and RCAR-IIWPG [14] protocols, with the exception of the driver position, where the hands were placed on the steering wheel as shown in Figure 1. The front seat measurements were performed in a mock-up of the front seat and steering wheel, as shown in Figure 1. The rear seat measurement was performed using the internal test procedures and settings of Volvo Cars, as described in Table 1; see Figure 2. BioRID II positioning was performed by specialists from Volvo Cars.

Before positioning the BioRID II in the front seat according to the ISO, RCAR and RCAR/IIWPG protocols, the SAE J826 manikin was used to set H-point and torso angles. After BioRID II *protocol* setup, the head restraints in the front and rear seats were removed. The calculations used to determine backset made use of the horizontal distance (X) between the 0-reference point (Figure 3) and the back of the volunteer's or the BioRID II head. The horizontal distance difference between the two measurement techniques (ISO, RCAR, RCAR-IIWPG protocols (B) and the 0-reference point (A) in Figure 3 used in this study) was 40 mm in the front seats and 25 mm in the rear seat, due to the design of the head restraint; this resulted in a BioRID II backset of 75 mm (40+35 mm) in the front seat and 100 mm (25+75 mm) in the rear seat; see Tables 2, 3 and Figure 3. This is important for further reading and understanding of the study data.

Results

BioRID II and human data: stature, weight and backset

Backset: female

For females, the mean backset was 39 mm in the driver position, 13 mm in the front passenger position and 98 mm in the rear passenger position. The BioRID II backset values (73 mm in the driver position, 76 mm in the front passenger position and 94 mm in the rear passenger position)

Figure 1. BioRID II, equipped with the measuring device, in the driver position.

corresponded to an 82nd percentile female in the driver position, a 95th percentile female in the front passenger position and a 41st percentile female in the rear passenger position; see Table 2.

Backset: male

For males, the mean backset was 81 mm in the driver position, 44 mm in the front passenger position and 109 mm in the rear passenger position. The BioRID II backset values (73 mm in the driver position, 76 mm in the front passenger position and 94 mm in the rear passenger position) corresponded to a 39th percentile male in the driver position, a 79th percentile male in the front passenger position and a 37th percentile male in the rear passenger position; see Table 3.

Discussion

The measurements performed in this study made use of a car which had achieved a good geometrical rating for its head restraint design in IIHS [6] testing. The interaction

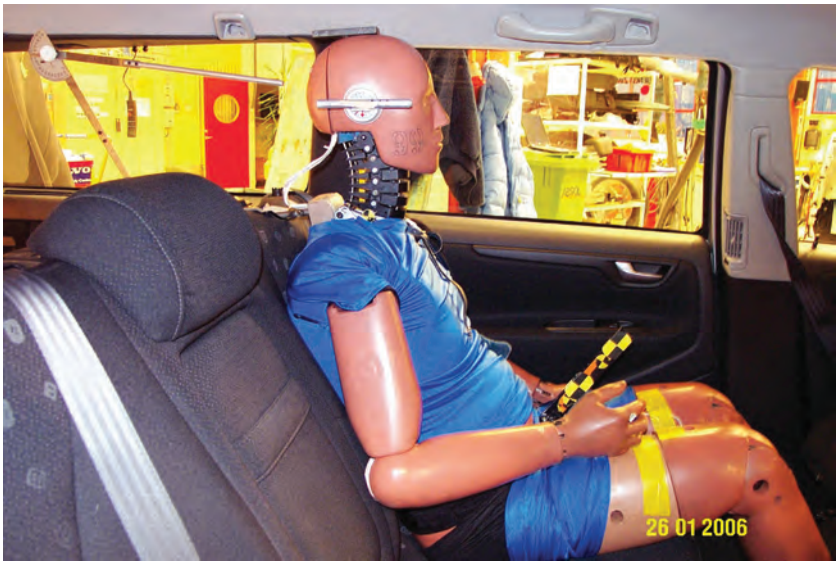

Figure 2. BioRID II, equipped with the measuring device, in the rear passenger position.

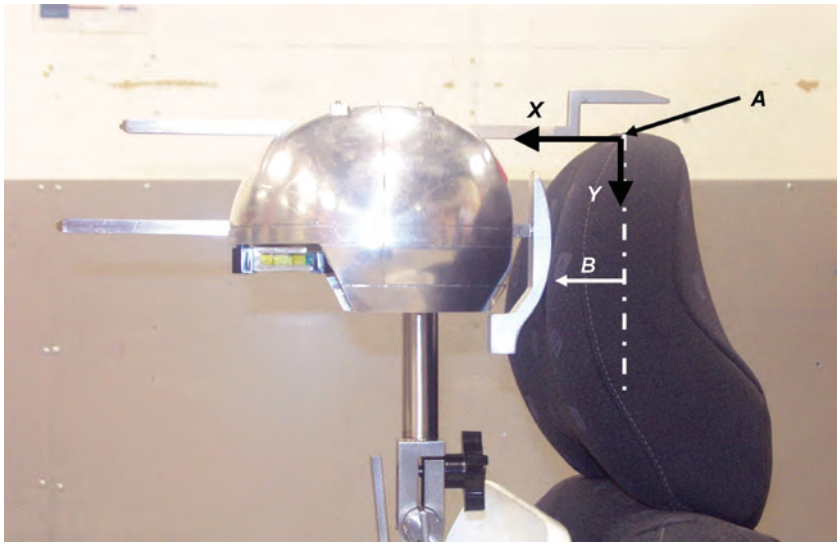

Figure 3. The H-point SAE J826 manikin with a head restraint measuring device. *A* denotes the 0-reference point for measuring backset on BioRID II and volunteers. *B* is the horizontal distance between an imaginary vertical line passing through the 0-reference point, and the most frontal aspect of the central part of the head restraint. The value of *B* was 40 mm in the front seat and 25 mm in the rear seat. *X* denotes the horizontal measuring direction, and *Y* the vertical measuring direction.

between human sizing data, seat/head restraint design and BioRID II geometrical design can be studied and evaluated, and if found to be of good quality, can serve as a model for other manufactures of seating systems.

The BioRID was made by a Swedish consortium to mimic the kinematics and kinetics of a human in rear end impact; at the time of its design and manufacture, no other dummy of this kind was available. The dummy design was based on available UMTRI anthropometry data, including information about stature, weight and sitting posture including back curvature. The anthropometry data used in the present study provide information on current occupant sizes of a Swedish population. These new data allow evaluation of how backset settings in volunteer seating positions differ from the standardised sitting positioning procedure of the BioRID II, and also contribute to showing how representative the BioRID II is in size.

Stature and weight

The anthropometry data in this study are quite representative for the Swedish population, especially as regards stature. The mean stature and weight of the volunteers were 179.6 cm and 86.9 kg for the males, and 166.8 cm and

Table 1. Positioning details for the H-point manikin SAE J826 and the BioRID II.

Settings	Front seat H-point manikin SAE J826	BioRID II	Rear seat H-point manikin SAE J826	BioRID II
Horizontal distance between back of head and head restraint (mm)	20	35	60	75
Torso angle (degrees)	25	25	30	30
Height (mm)	24	NA	85	NA
Pelvic angle (degrees)	NA	26.5	NA	31.5
Seat motor settings				
Motor 1: Length adjustment (revolution from rearward 0-starting point = mid range)	755	755	Fixed	Fixed
Motor 2: Seat back angle adjuster (revolution from rearward 0-starting point)	1613	1613	Fixed	Fixed
Motor 3: Rear seat pan height adjustment (revolution from from rearward 0-starting point = mid range)	252	252	Fixed	Fixed
Motor 4: Front seat pan height adjustment (revolution from rearward 0-starting point = mid range)	192	192	Fixed	Fixed

NA = not available.

71.1 kg for the females. In comparison, stature and weight for the whole Swedish population (16–84 years) are 179.5 cm and 82.4 kg for males, and 165.5 cm and 66.6 kg for females SCB [15]. Local survey data from Västernorrland County Council [18] show that males are 1.2 kg heavier and 1.3 cm shorter, and females are 2.4 kg heavier and 0.5 cm shorter in the local community than shown by national SCB data [15]. This explains some of the weight difference (+4.5 kg) between local and national Swedish data. UMTRI data by Schneider [16] from the HANES survey, conducted between 1971 and 1974 in the United States for the 18–74 age group, showed stature and weight of 175.3 cm and 77.1 kg for males, and 161.5 cm and 62.1 kg for females. Thus, for both genders, the stature and weight differences between the United States and Sweden over slightly more than 30 years are about 4 cm and 5 kg, respectively.

As shown in Tables 2 and 3, the 50th percentile male BioRID II corresponds to a 35th–45th percentile male in stature and a 35th percentile male in weight, and to a 96th percentile female in stature and a 69th percentile female in weight. The BioRID II is 11 cm taller and 8 kg heavier than a 50th percentile female. Swedish females are taller than those in the United States, with a stature of 165.5 cm, compared to 162.5 cm for US females [11]. Mean female stature varies in other countries; the mean stature of females in 10 European countries is 163.7 cm [2], that of Chinese females is 154.5 cm Bell [1] and that of Brazilian females is 160.3 cm [5]. Dutch and Norwegian females are slightly taller than Swedish females [2]. The sitting male and female data determined by the present study could provide a valuable input to the design of a possible next member of the BioRID family, which thus could be made to represent a larger part of the female population.

Backset

The most obvious finding in this study was the difference in backset between a driver with hands on the steering wheel and a front seat passenger with hands resting in lap. In both genders, the mean backset for males in the front passenger position was roughly half that for the driver position, and the corresponding data for females was 1/3. Backset increased for both males (37 mm) and females (26 mm) when volunteers held their hands on the steering wheel (driver position), in comparison to the front seat passenger posture; no other postural changes were made. In volunteers, this posture change was mainly produced by increased kyphosis in the thoracic spine.

Table 2. Female volunteer data for stature (cm), weight (kg) and backset (mm), and corresponding Bio-RID II data. Female data are presented in 5-percentile steps. Backset is defined as the horizontal distance between the back of the head and the front of the seam on top of the head restraint.

BioRID II	Stature (cm)	Weight (kg)	Driver Backset (mm)	Front seat passenger Backset (mm)	Rear seat passenger Backset (mm)
	178	78	73	76	94
Females $n = 76$	Stature (cm)	Weight (kg)	Backset (mm)	Backset (mm)	Backset (mm)
Median	167	70	41	10	100
Mean	167	71	39	13	98
SD	7	13	36	31	26
Range	152	48	−29 to 117	−46 to 95	36 to 197
Female Percentile					
5	156	51	−18	−36	58
10	158	54	−7	−23	64
15	160	58	−1	−19	68
20	160	60	3	−15	72
25	163	62	9	−9	80
30	163	63	13	−7	82
35	163	65	20	−4	88
40	165	65	32	5	93
45	167	68	37	7	96
50	167	70	41	10	100
55	168	71	43	14	101
60	168	74	50	17	104
65	170	75	56	21	109
70	170	79	61	27	111
75	171	80	64	32	112
80	172	82	69	41	115
85	174	85	79	46	118
90	175	90	89	58	136
95	177	95	102	73	150

Table 3. Male volunteer data for stature (cm), weight (kg) and backset (mm), and corresponding Bio-RID II data. Male data are presented in 5-percentile steps. Backset is defined as the horizontal distance between the back of the head and the front of the seam on top of the head restraint.

BioRID II	Stature (cm)	Weight (kg)	Driver Backset (mm)	Front seat passenger Backset (mm)	Rear seat passenger Backset (mm)
	178	78	73	76	94
Males $n = 78$	Stature (cm)	Weight (kg)	Backset (mm)	Backset (mm)	Backset (mm)
Median	180	85	82	45	107
Mean	180	87	81	44	109
SD	6	16	44	35	31
Range	167	63	−5 to 209	−27 to 152	50 to 224
Male percentile					
5	168	67	9	−12	62
10	172	68	17	−7	71
15	173	71	38	3	77
20	173	73	45	14	81
25	175	74	47	20	89
30	176	75	55	25	90
35	178	78	69	29	93
40	178	81	76	35	96
45	178	82	78	39	99
50	180	85	82	45	107
55	180	86	86	48	111
60	181	89	89	51	118
65	183	90	92	56	121
70	183	92	104	61	124
75	184	96	108	67	128
80	184	98	117	78	134
85	185	105	128	86	141
90	187	113	146	90	144
95	190	118	149	102	161

The standardised seating procedure of the BioRID II does not differentiate between the driver and passenger position, and the current design of the BioRID II does not automatically adjust the backset when changing the arm position. The results of the present study show that this standardised seating procedure more closely resembles the driver position than the front seat passenger position. In comparison to the BioRID II, a 50th percentile male had an 8-mm longer backset in the driver position, and a 32-mm smaller backset in the front passenger position, while a 50th percentile female had a 32-mm smaller backset in the driver position and a 66-mm smaller backset in the front passenger position. The backset differences between the BioRID II and the volunteers were smaller in the rear passenger position.

There were also differences between the males and the females with regard to backset. Females have lower stature, and 50–70 mm shorter arms, than males [12]. This may explain why females chose different seat adjustments than males. Females sit with more upright back support, 3° different from males [9]. Three degrees gives a 30–35 mm less horizontal distance at the level of the back of the occupant's head. These data might explain why females had lower backset than the males in the front seat positions.

Seat adjustments

Table 4 shows both males and female to sit 61 and 20 mm further away from the steering wheel in comparison to the BioRID setting. Back rest angles for males and females are

Table 4. Seat adjustment settings. Mean values in mm and degrees for males and females and standard deviation (SD), and BioRID setting.

Seat adjustment	BioRID	Male	SD	Female	SD
Length adjustment in (mm), 0-value is max. rearward position	140	79	44	120	46
Back rest angle in (degrees), 0-value is max. reclined position of back rest	42	43	6	46	5
Height rear seat cushion in (mm), 0-value is the max. lowest position	21	15	13	19	14
Height front seat cushion (mm), 0-value is the max. lowest position	15	13	10	16	10

slightly more upright, 1 to 4°. Both these settings affect backset. A 1° change in the back rest angle is an approximately 1 cm change of the horizontal distance of the top head restraint level.

Other findings and observations

The volunteers in this study adjusted the front seats without having the head restraint in place. This resulted in an interesting ergonomic finding for both genders. Females with lower stature, in particular, showed negative (−) backset values (driver <160 cm and front passenger position <164 cm), indicating a possible conflict between ergonomic comfort preferences and good geometric safety design. Negative backset values were also seen for males of <173 cm in the front passenger position.

In order to evaluate differences between drivers and passengers, improvements could be made to the BioRID II seating procedure to better reflect these differences in backset. Additionally, in future development of the BioRID II, it could be of interest to see a technical feature allowing an easy adjustment of the thoracic spine shape from kyphosis to an erect posture without affecting the eye position of the manikin; this is not easily achievable in the current BioRID II.

To sum up, the contributions of this study include detailed measurements of seated car occupants, providing knowledge of how representative the size and seating position of the BioRID II dummy are, as well as input to potential further improvements with respect to both dummy development and dummy seating procedures such as the ISO [7], RCAR [13] and RCAR-IIWPG [14] protocols.

Conclusions

Human backset differed between the driver position and front seat passenger position for both genders; this difference is not reflected by the BioRID II using the standardised seating positioning procedure. Mean backset for males in the front passenger position was roughly half that for the driver position and the corresponding data for females was only 1/3. In comparison to a *local* Swedish population of female and male volunteers, sitting as drivers (hands on steering wheels), front seat passengers (hands in lap) and rear seat passengers, the BioRID II, when positioned according to the ISO [7], RCAR [13] and RCAR-IIWPG [14] protocols, corresponds in stature to a 35th–45th percentile male and a 96th percentile female, and in weight to a 35th percentile male and a 69th percentile female.

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Influence of Gender, Height, Weight, Age, Seated Position and Collision Site related to Neck Pain Symptoms in Rear End Impacts

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Abstract Rear end vehicle collisions can result in occupants suffering neck pain symptoms of varying degree and duration. These injuries are generally called whiplash injuries and they are common and costly. This study analyses the occurance and duration of neck pain symptoms of one particular vehicle make with focus on the influence of occupant specific information. Data collected from a Swedish vehicle make, model year 1993 up to model year 2007 at a maximum of three years old, were analysed.

The results from this study show that passengers are more likely than drivers to suffer neck pain symptoms, in crashes that occurred in that particular make of car. No significant differences in risk related to age, gender weight, and height could be identified, except for: Females aged 35-44 had higher risk to have long and medium term neck pain symptoms than males in the same group of age. Males aged >=65 had higher risk to have long and medium term neck pain symptoms than males aged 35-44. Females in group “Braking” had higher risk of any type of neck pain symptoms than males.

Where the occupant was seated in the front seat of the carinfluenced the occurance of neck pain symptoms and their duration for both males and females, with passengers posing a higher risk of suffering neck pain symptoms compared to drivers. Of the drivers, 17 percent reported neck pain symptoms compared to 44 percent of the passengers. When grouped into the categories males and females, 15 percent of the male and 19 percent of the female drivers reported neck pain symptoms compared to 44 percent of the male and 43 percent of the female passengers. With respect to the different collision sites, rear end collisions at traffic lights most often resulted in occupants reporting neck pain symptoms. Collisions in roundabouts most often resulted in different impact scenarios and occupants reported suffering neck pain symptoms of mid and long term duration.

The result of this study indicates the need for improved understanding of the differences between driver and passenger response in different driving scenarios. In addition, occupant charactersisics should also be studied.

Keywords Neck pain symptoms, Rear impact, Seat, Injury protection

I. INTRODUCTION

Neck pain symptoms, of varying degree and duration sustained in low severity vehicle collisions, commonly denoted whiplash injury, are a worldwide problem. Such injuries are costly since they are frequent and can lead to long lasting pain and disability. In Europe alone, the annual cost for whiplash injuries has been estimated to be 10 billion Euros [1]. Whiplash injuries account for ~70 percent of all crash-related injuries leading to disability [2]. The majority of those who experience initial neck symptoms following a car crash recover within a few weeks or months of the crash [3]. However, 5–10 percent of individuals will experience permanent disabilities of varying degrees [3–5]. These injuries occur at relatively low speed changes (typically <25 km/h) [6–7] in impacts from all directions. Rear impacts, however, featured most frequently in the injury statistics [8].

Since the mid-1960´s, statistical data have shown that females have a higher risk of sustaining whiplash

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injuries than males, even in similar crash conditions [9–18]. According to these studies, the whiplash injury risk is up to three times higher for the females compared to the males.

Different seat designs and whiplash protection systems have shown different degrees of protection against such injuries [19]. Furthermore, the whiplash injury risk is dependent on in which seat the occupant is positioned. Some studies have found that the whiplash injury risk is greater in the front passenger seat compared to the driver seat [13][20]. Other studies have reported that the whiplash injury risk is greater in the driver seat compared to the front passenger seat [21–22]. Several studies have indicated that front seat occupants have a higher whiplash injury risk than rear seat occupants [20][22–24]; however, when looking at the long term injuries, [17] found a different correlation for the females. In that study a paired comparison was performed on all neck injuries reported to the Swedish insurance company Folksam, following rear impacts during 1990–1999.

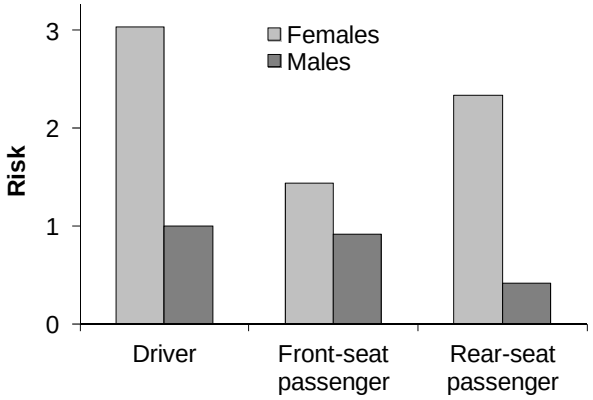

Fig. 1. The risk of permanent whiplash injury in relation to the male driver risk (normalised to 1) for different seating positions in rear impacts. Based on [17].

The males had a lower injury risk in the rear seat compared to the front seat, while the females had a considerably higher injury risk in the rear seat; the lowest risk for the females was found for the front passenger seat (**Fig. 1**). The risk of permanent disability was three times higher for female drivers compared to male drivers. Similarly, permanent disability was 1.5 times higher for female front seat passengers and more than five times higher for female rear seat passengers.

The objective of this study was to evaluate the influence of front seat seating position, crash location, weight, height, age and gender on the risk of neck pain symptoms in a rear collision.

II. METHODS

Description of the study

Data collected from the particular Swedish vehicle make, model year 1993 up to model year 2007 at a maximum of three years old, were analysed. The dataset contained single event rear end impacts resulting in repair claims containing occupant specific data of both uninjured and injured occupants. A questionnaire was sent out to the owner of the car, requesting information about occupant age, weight,height, seatbelt usage, etc. The data were analysed to study the influence of gender, height, weight, age and seating position in relation to duration of neck pain symptoms.

All new vehicles sold in Sweden, by the vehicle make analysed in this study, is covered by a three year warranty. During this period, any collision requiring repair is covered by the warranty. The insurance company collected data from all rear end collisions in model year 1993 up to model year 2007 vehicles[1], at a maximum of three years old at collision date. A questionnaire was sent to the owner of the vehicle collecting additional information about the persons involved in the crash. Some collisions were found to be outside the scope of this study. Cases excluded were multiple event collisions, parking lot accidents with no occupants in the struck vehicle, collisions involving a motorcycle or moped and duplicate cases. This resulted in 1,142 cases of rear end collisions involving 1,345 individuals. Information from accident reports, repair valuations, pictures of the struck vehicle and police reports was available. However, all categories of information were not available for all cases. In some cases, only an accident report or repair valuation was available, whereas in other cases, different combinations of the above information were available.

The dataset consisted of two different collection periods. Survey 1 was performed for vehicles with model year 1993 up to 1999 and Survey 2 with model year 2000 up to 2007. Some of the approaches differed between the two surveys and are described below.

[1] Saab 9-3, model year <2003 and Saab 9-5 are equipped with the SAHR gen I whiplash protection system. Saab 9-3, model year ≥2003 are equipped with the SAHR gen II whiplash protection system.

Survey 1 & 2: Police reports and additional information gave a detailed view of the crash circumstances and course of events. The accident reports contained information on individuals or at least the drivers involved, crash circumstances and injury outcome. Survey 2 only: Repair valuations included repair cost and parts repaired or replaced. Pictures of the struck vehicle gave increased understanding of the crash severity.

Survey 1: The change of velocity (Δv) was estimated based on comparison of pictures from crash-tested vehicles in the laboratory, i.e., deformations were compared cars crashed on the field with the cars that had been crashed in the crash-lab where Δv was documented.

Survey 2: Crash severity was classified based on information from the repair valuation. A damage severity scale from 1 to 5 was developed:

Category 1: lacquer and paint damage, sheet-metal dents, renewal of plastic details such as brackets for parking sensors as well as repair or replacement of the rear bumper.

Category 2: the bumper foam blocks had been renewed.

Category 3: the towing hook had been repaired or replaced.

Category 4: constructional beams had been repaired or replaced.

Category 5: the struck vehicle was in such bad shape that it had to be towed from the accident.

To obtain additional information about occupant height and weight, usage of seatbelt, etc, a questionnaire was sent by post to the owner of the vehicle and in some cases telephone interviews were conducted. Survey 1 was performed for vehicles with model year 1993 up to 1999 and Survey 2 model year 2000 up to 2007. The questionnaires were posted as follows:

- If any injuries were claimed in the accident report (Survey 1 & 2)
- If damage severity was classified as a 4 or 5, whether neck pain symptoms were reported or not (Survey 2)
- If damage severity was classified as a 3 and the vehicle was fitted with a towing hook, whether neck pain symptoms were reported or not (Survey 2)

In cases where the relevant individuals could not be reached, the duration of their neck pain symptom status was considered unknown. If a questionnaire had not been posted and passenger information was not available in the accident reports, the driver was assumed to be the sole occupant of the car.
Below is outline of how the occupants´ neck pain symptoms were categorised into four different categories, or into the category unknown or no neck pain symptoms:

NI: No neck pain symptoms
ST: Short Term neck pain symptoms (neck pain lasting up to one week)
MT: Medium Term neck pain symptoms (neck pain lasting more than one week but less than ten weeks)
LT: Long Term neck pain symptoms (neck pain lasting longer than ten weeks)
INJ: Only in Survey 1: If they noted some kind of neck pain in the injury report, but had not answered the questionnaire
N/A: Unknown status (information unavailable)

Description of the data set

The dataset contained information about 1,345 individuals, mostly from Survey 2. Rear seat occupants below the age of 16 were removed from the dataset. **Table I** 'N red' shows the number of cases used for analysis in this study.

TABLE I
NUMBER OF OCCUPANTS INVOLVED IN REAR END COLLISION

	Number (N)	Number(N red)[a]
Survey 1	203	180
Survey 2	1,142	890
Total	1,345	**1,070**

[a] Occupants: only driver, front-seat passenger and aged 16 and older.

The response rate was approximately 60 percent from both surveys. In total, there were 223 responses from

different households with information on 360 occupants. Of these occupants there were 303 individuals who satisfied the requirements (**Table II**, 'Occupants (red)').

TABLE II

SUMMARY OF THE TWO SURVEYS:NUMBER OF SENT QUESTIONAIRS, RESPONSES

	Posted	Responses	Response rate	Occupants	Occupants (red)
Survey 1	99	57	58%	85	78
Survey 2	275	166	60%	275	225
Total	374	223	60%	360	**303**

In **Table III** the total number of occupants is presented. In some cases data were missing, denoted N/A.

TABLE III

THE GENDER, AGE, WEIGHT, HEIGHT AND THE DURATION OF NECK PAIN SYMPTOMS

	Background details				Injury details (*total sum=1,273*)					
	Gender (n)	Age (n)	Weight (n)	Height (n)	NI (n)	ST (n)	MT (n)	LT (n)	INJ (n)	N/A (n)
Survey 1	**185**	**185**	**78**	**78**	**101**	**16**	**6**	**13**	**44**	**5**
Driver	**141**	**141**	**58**	**58**	**83**	**11**	**5**	**10**	**27**	**5**
Female	19	19	8	8	10	2		1	6	
Male	115	113	49	50	71	9	5	9	21	
N/A	7	9	1		2					5
Passenger	**44**	**44**	**20**	**20**	**18**	**5**	**1**	**3**	**17**	
Female	30	28	14	16	11	2	1	3	13	
Male	8	7	4	4	1	3			4	
N/A	6	9	2		6					
Survey 2	**1,088**	**1,088**	**209**	**209**	**774**	**74**	**22**	**20**	**0**	**198**
Driver	**1,006**	**1,006**	**164**	**164**	**735**	**52**	**18**	**17**		**184**
Female	150	143	34	36	98	11	5	7		29
Male	597	570	123	122	424	41	12	10		110
N/A	259	293	7	6	213		1			45
Passenger	**82**	**82**	**61**	**61**	**39**	**22**	**4**	**3**		**14**
Female	45	40	33	32	23	17	2	1		2
Male	31	24	19	19	14	5	2	2		8
N/A	6	18	9	10	2					4
Total	**1,273**	**1,273**	**303**	**303**	**875**	**90**	**28**	**33**	**44**	**203**

Table IV shows that mean values of age were not significantly different in the dataset. The males were 17.8 heavier and 13.7 cm taller than females.

TABLE IV

MEAN VALUES OF AGE, WEIGHT AND HEIGHT

	Age	Weight (kg)	Height (cm)
Females	45.4	65.4	166.5
Males	45.9	83.2	180.2
Difference	**0.5**	**17.8**	**13.7**

Statistical analysis

Age, weight and height were divided into groups containing intervals of 10 due to if less intervals were selected, i.e., intervals of 5, the the number of observations in the dataset was too few in each group. Due to the low number of observations, predominantly in the group LT+MT, it was not possible to use the Chi-Square Test in this case. When performing this test, the expected values in separate cells must be >=5, which was mostly not the case. Therefore, Fisher's Exact Test [35] was used instead, however, this test can only manage a

2x2-matrix. Therefore NI and ST were merged in order to obtain differences of NI +ST vs. MT+LT (P-values=p, if p<.05 = significant) when using the Fisher's Exact Test. The paired combinations were only conducted for groups greater than a minimum 20 observations.

III. RESULTS

All cases contained information about gender, age, collision action, and collision site. Information about tow hook, weight, height and (Body Mass Index) BMI were collected for the cases of higher impact severity since the questionnaire for the major study (Survey 2) was sent to occupants in severe crashes. LT and MT were merged in all figures in order to reduce uncertainty due to the low number of observations. In most cases there were too few observations of passengers and therefore this group will not be presented due to its unreliability. If a group illustrated as a bar in the Figures consist of <20 observations, the bar have dotted lines, to illustrate the uncertainty due to the few observations in this group. All figures in the result section have tables of statistics including p-values in the Appendix.

Driver–Passenger: Gender

Of the passengers, 44 percent had reported some kind of neck pain symptoms in comparison to the drivers for which 17 percent had reported neck pain symptoms (**Fig. 2**). In **Fig. 2** drivers and front-seat passengers are divided into the categories Males and Females. There were marginal differences between males and females. Statistical significance tests confirm that there were no differences between the genders (p_{Dr}=.182, p_{Pa}=.733). For male drivers, the tendency to have LT or MT was lower than for female drivers. Male drivers and male passengers showed a relatively major difference of LT and MT (8%), but the statistical test (p=.094) indicates that the difference is not significant. Female drivers and passengers had similar amount of LT + MT symptoms; however, female passengers compared to female drivers had considerably more short-term neck pain symptoms. The male passengers had more short-term neck pain symptoms than the male drivers, equivalent to female passengers.

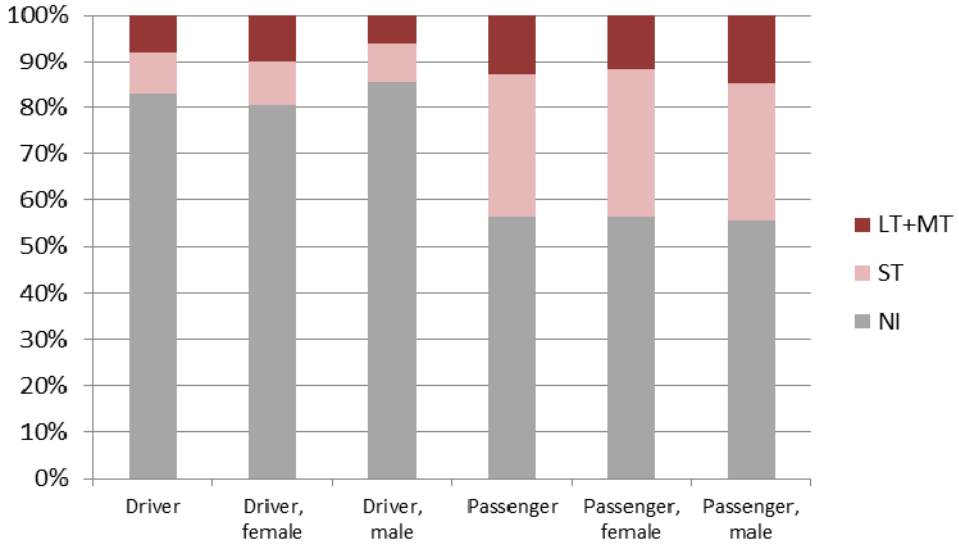

Fig. 2. The duration of neck pain symptoms, occupant front seat position and gender. (n_{dr}=715, n_{pa}=87)

Regarding severity of impact, a bias was found in the data showing slightly higher severity of the cases involving passengers.

Driver: Tow hook and Gender

There were no statistically significant results, but the it was apparent that the tendency for female drivers to have increased risk of medium and long term neck pain symptoms if the vehicle was fitted with a tow hook (**Fig 3**).

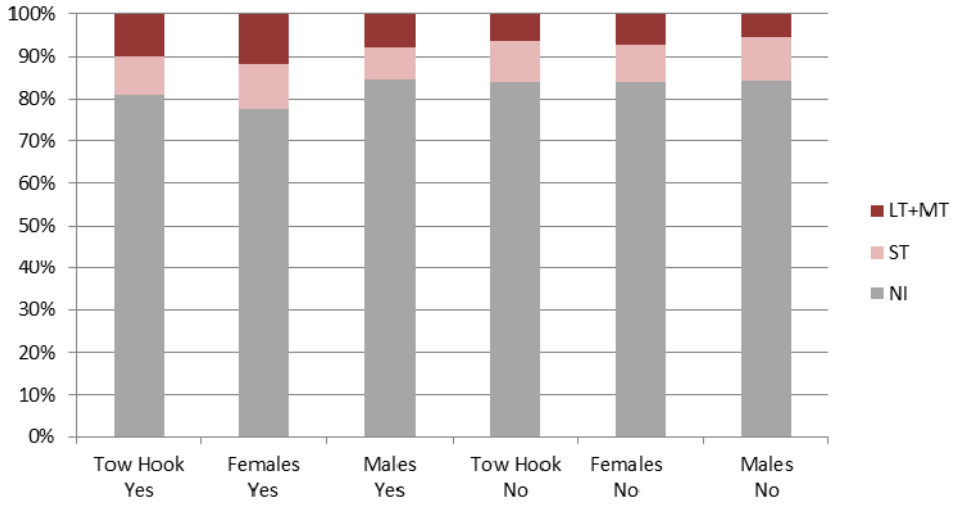

Fig. 3. The duration of neck pain symptoms of drivers with (yes) and without (no) tow hook, and gender. (n_{no}=293, n_{yes}=406)

Driver: Age and Gender

What influence age of the occupants has, is shown in **Fig 4 and 5**. Females, aged 35-44, had the highest risk of medium and long term neck pain symptoms, males in this group had the lowest risk. Fisher's Exact Test confirms that the differences are significant. Males, aged >=65 have higher risk of having LT and MT compared to the other age groups for males (significant between 35-44, p=.049). The observations for females, ages 16–34 and >=55, were too few to draw any conclusions from, for males the number of observations were too few for the ages 16-24.

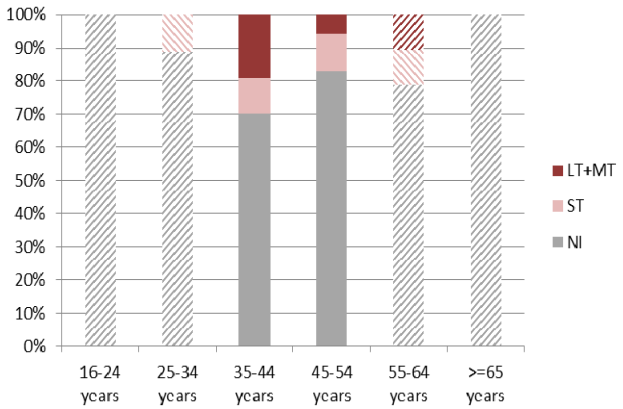

Fig. 4. The duration of neck pain symptoms, FEMALE distributed in age groups. (n=127)

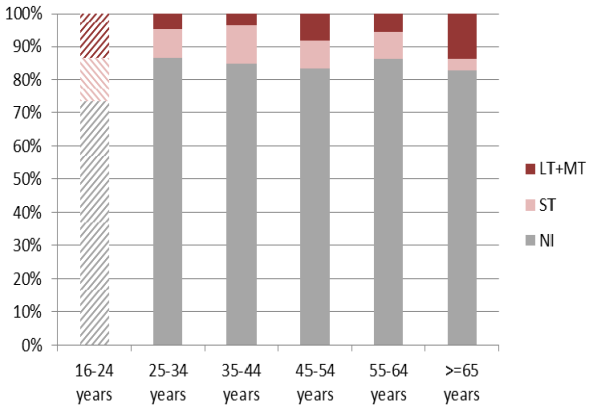

Fig. 5. The duration of neck pain symptoms, MALE distributed in age groups. (n=557)

Driver: Weight and Gender

The influence of weight of the occupants is shown in **Fig. 6-7**. Only weight group 60–69 (n=20) kg for the females had sufficient number of observations from which to draw any conclusions. (**Fig. 6**). The tendency was that males in weight groups 80–89 and 90–99 kg had decreased risk of MT and LT (**Fig. 7**). Due to the low number of cases in groups 60–69 kg (n=6) and 110 and higher (n=5), conclusions could not be drawn about these weight groups.

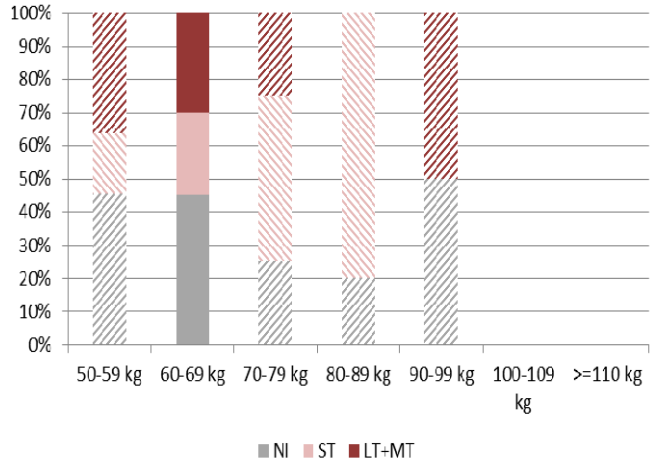

Fig. 6. The duration of neck pain symptoms, FEMALE distributed in weight groups. (n=42)

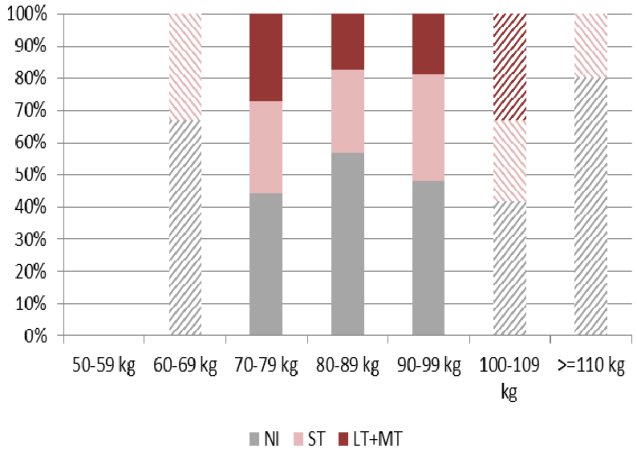

Fig. 7. The duration of neck pain symptoms, MALE distributed in weight groups. (n=167)

Driver: Height and Gender

The female height groups 160–169 (n=27) had sufficient number of observations in order to identify trends (**Fig. 8**). **Fig. 9** had too few observations in height groups 150–159 cm (n=1), 160–169 cm (n=4) and >=190 cm (n=16). Therefore conclusions could not be drawn about them. There was a tendency for males in height group 170–179 cm compared to 180-189 cm to have an increased number of MT and LT, but there were too few observations to have a significant value.

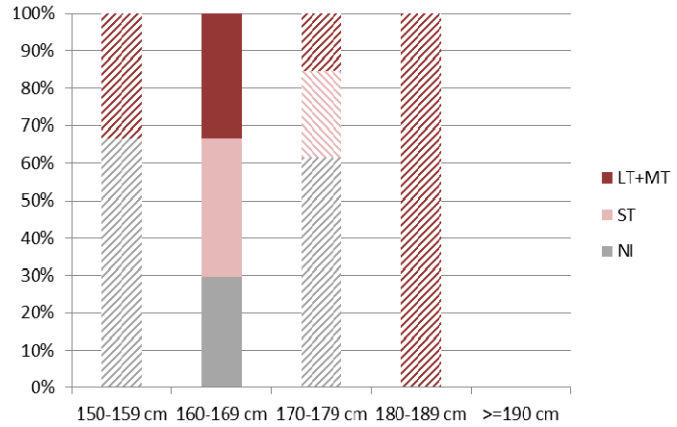

Fig. 8. The duration of neck pain symptoms, FEMALE distributed in height groups. (n=44)

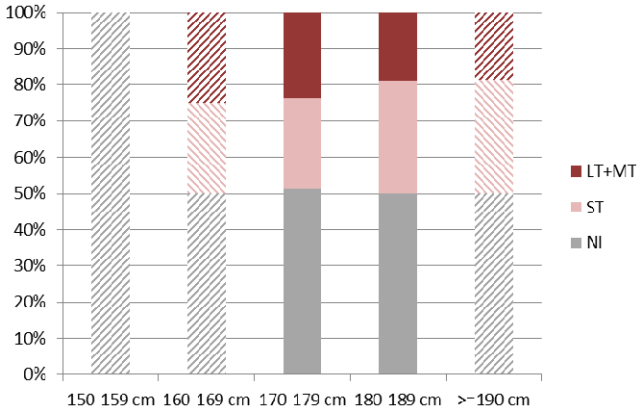

Fig. 9. The duration of neck pain symptoms, MALE distributed in height groups. (n=167)

Driver: BMI and Gender

BMI were divided into three different groups, persons of average weight (18.5 - <25), overweight persons (25 - >30) and obese persons (>=30). None of the persons in the surveys were underweight. There were approximately 30 percent females of average weight in the group who had medium and long term neck pain symptoms, compared to males in the same BMI group who had a 10 percent lower value. However, this is not significant (p=.310). Male-groups 18.5- >25 and 25- <30 have no statistical significance (p=1.000).

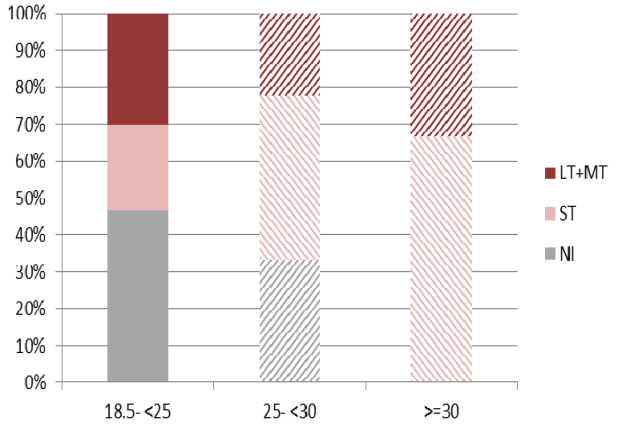

Fig. 10. The duration of neck pain symptoms, FEMALE distributed in BMI groups. (n=42)

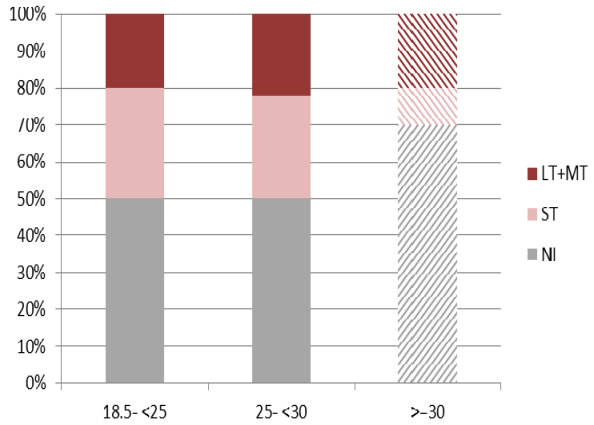

Fig. 11. The duration of neck pain symptoms, MALE distributed in BMI groups. (n=166)

Driver: Collision Action and Gender

Collision Action was divided into three groups; if the vehicle was braking, driving or standing still. These results are based on cases from Survey 2. In the Braking group, females reported more LT and MT than males and it proved significant (p=.003) (**Fig. 12**). With regards to the Driving group, almost all were uninjured irrespective of gender; however this group comprised too few females in this group. For the Collision Action Stationary group, there were minor differences between the genders, not significant (p=0.549) (**Fig. 12**).

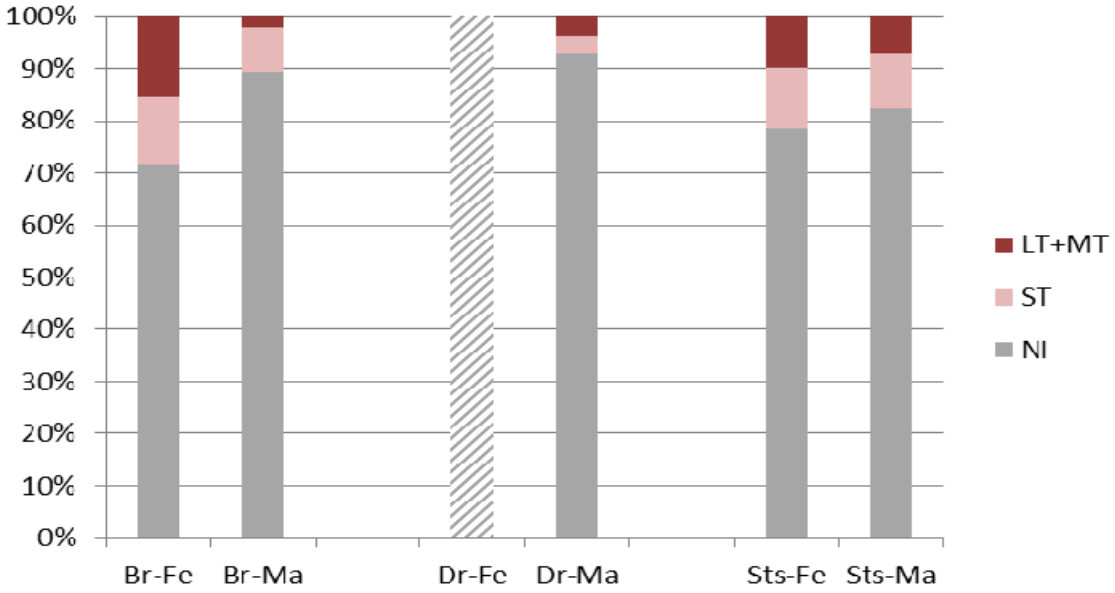

Fig. 12. The duration of neck pain symptoms, gender distributed in Collision Action (Br-Fe = Braking, female; Br-Ma= Braking, male; Dr-Fe= Driving, female; Dr-Ma= Driving, male; Sts-Fe= Standing still, female; Sts-Ma= Standing still, male). (n_{Br}=188, n_{Dr}=35, n_{Sts}=274)

Driver: Collision Site

The Collision Site group was divided into seven main groups; Traffic light, Queue, Zebra crossing, Give way, Roundabout, Give way roundabout, Stop sign and Other. These results are based on cases from Survey 2. The most common place for collisions to occur was at Traffic lights (22 percent), secondly Queues (19 percent), thirdly Zebra crossings (17 percent) and in fourth place at Give way signs (16 percent) (**Fig. 13**).

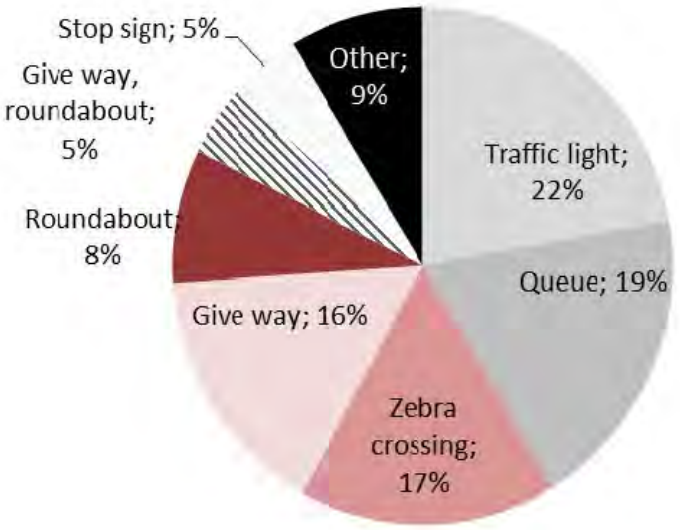

Fig. 13. Most common places for collisions to occur. (n=263)

In the Roundabout group, the drivers had the highest risk (13 percent) of sustaining LT and MT. The lowest risk for drivers of sustaining LT and MT was at the Give way and Stop signs out of the Collision Site groups. Of the four major groups, Give way was the only group with no LT or MT cases (**Fig. 14**). Significant difference were found between the Give way and Roundabout groups, p=.039.

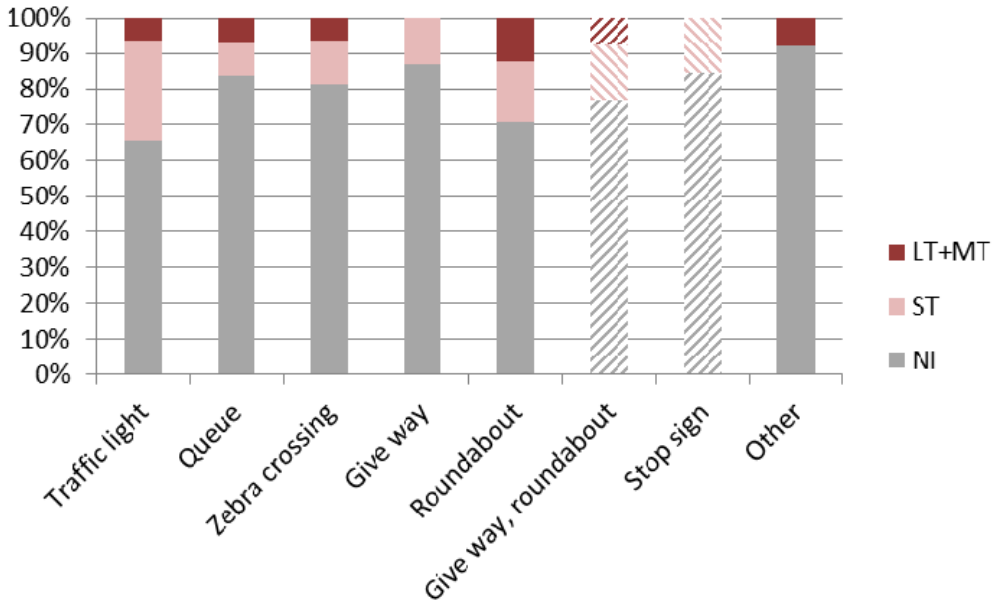

Fig. 14. The duration of neck pain symptoms and Collision Site. (n=228)

IV. DISCUSSION

Male passengers seated in the front seat tended to have a greater risk of sustaining LT+MT neck pain symptoms (15 percent) compared to male drivers (6 percent), while the females had a similar risk in either front seat (drivers: 10 percent, front seat passengers: 12 percent) (**Fig. 2**). Greater differences between the two front seats were recorded for the ST neck pain symptoms for the males as well as for the females (drivers: ~10 percent, front seat passengers: ~30 percent). The result from this study is supported by earlier studies [13][20], however, other studies have reported that the whiplash injury risk is greater in the driver seat compared to the front passenger seat [21–22]. The long-term whiplash injury risk was reported to be approximately twice as high for female drivers compared to female front seat passengers in rear impacts [17] (**Fig. 1**). Factors that may contribute to these differences could be seated posture and grip of the steering wheel. Little is known about the

influence of the steering wheel with regard to the dynamic response of the occupant during the seatback interaction and/or during the rebound in rear impacts. In frontal impacts, an increased risk of whiplash injury symptoms was reported for occupants tightly gripping the steering wheel or straightening their arms [25]. In addition, it may be that different anti-whiplash systems and seat designs influence the distribution of risk between the passenger and driver position differently. Furthermore, in-depth comparison between studies in the literature might show differences in distribution of, for example, impact severity which could influence the results.

The LT+MT neck pain symptoms risk tended (not statistically significant) to be greater in vehicles fitted with a tow bar compared to vehicles lacking a tow bar (**Fig. 3**). This tendency was more pronounced for the female drivers (12 percent versus 7 percent) compared to the male drivers (8 percent versus 5 percent). Similar results were reported by [26], reporting a 22 percent higher long term whiplash injury risk in rear impacts sustained in cars fitted with a towing hook compared to cars without a towing hook (same car model) [26]; the data were however not separated for females and males. The presence of a towing hook may increase the severity of the crash pulse, which has been shown to influence the injury risk. [7][27].

For female drivers, the LT+MT neck pain symptoms risk were significant higher than for the males for the age group 35-44 years, while for the male drivers the risk was significantly higher than for the females in the age group >=65 years (**Fig. 4**). At older ages the risks were lower for both females and males. Similar trends have been reported in earlier studies, indicating that the whiplash injury risk seems to peak at younger/middle ages and decrease at older ages [15][22][28]. The reason may possibly be that increasing age is associated with increasing stiffness of the neck. For example, [29] found that the Active Range of Motion of neck extension was greatest for young people and decreasing as the age increased.

The LT+MT neck pain symptoms tended to be greater for either very light or for very heavy drivers; this tendency was noted for both males and females (**Fig. 5–6**). In earlier studies the weight of the occupants appeared to have minor/no correlation with the whiplash injury risk [15][23][30]. The LT+MT neck pain symptoms tended to decrease for increasing height of both male and female drivers (**Fig. 7–8**). These results deviate from earlier studies, reporting generally increasing trends for increasing height for males and females [10][15][21–22]. In addition, [25] showed an increasing trend of risk for increasing height for male occupants, while for female occupants the risk was approximately the same for all the selected height intervals. The influence of height may be different for different types of seats. The main contributing factors related to height and occurrence of neck pain symptoms are still to be established.

The LT+MT neck pain symptoms were considerably less for male drivers (2 percent) compared to female drivers (15 percent) if the vehicle was impacted while braking (**Fig. 9**). Applying the brakes of the vehicle may, due to the momentum of the upper body, have resulted in the head restraint (HR) distance being increased at the time of the impact. For this reason it is possible that the females were affected to a greater extent by the braking event compared to the males. Based on volunteer tests, it was found that female volunteers subjected to an unexpected braking event seemed to experience a longer forward motion than males of the same size [31]. Furthermore, males and females of the same seated stature appeared to have a different motion pattern in driver as well as passenger positions. The forward motion of the male volunteers seemed to follow the deceleration pulse, with only a short time at the turning point, whereas the females stayed in the most forward position for a longer period, until the brake released [31]. A long HR distance is associated with increased whiplash injury risk [4][24–25][28][32–33]. Moreover, [28] concluded that “Not only are women more likely than men to suffer neck injuries in rear impacts, but they are affected more by changes in head restraint positioning”. In addition, [34] found that improved head restraint geometry reduces the whiplash injury risk to a greater extent for females than for males.

The LT+MT neck pain symptoms for drivers tended to be greater in roundabouts compared to traffic lights. The reason may be that drivers, to a greater degree, may be out of position and have their heads’ turned at the time of the impact in a roundabout compared to at a traffic light. The question may be raised whether the roundabout is an environment with higher risk compared to traffic lights with regards to long term neck pain symptoms.

The low number of observations in this study limited the posibilities to draw conclusions for several of the subgroups. A larger dataset would be required in order to establish whether trends seen in this study are stastistically significant findings. In particular, it was not possible to relatethe influence of impact severity any

particular subgroup. It may be that some collision sites are designed such that more severe crashes in terms of neck pains symptoms, are generated at these sites. A larger dataset may thus give new insights into how the road system could be improved. Furthermore, the results of this study indicate the need for improved understanding of the differences between driver and passenger performance in different driving scenarios. In addition, how the head to head restraint distance is influenced during braking and how it affects the injury outcome should be studied for both males and females.

V. CONLUSIONS

The results show that passengers are more likely to suffer neck pain symptoms than drivers in the collisions that occurred in the same make of a particular car. No significant differences in risk related to age, gender weight, and height could be identified, except for:

- Females aged 35-44 had higher risk to have long and medium term neck pain symptoms than males in the same group of age.
- Males aged >=65 had higher risk to have long and medium term neck pain symptoms than males aged 35-44.
- Females in group "Braking" had higher risk of any type of neck pain symptoms than males.

Regarding the collision site, rear end collisions that occurred at traffic lights most often resulted in neck pain symptoms. Collisions in roundabouts was the most common reason for mid and long term neck pain symptoms.

VI. ACKNOWLEDGEMENT

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VIII. APPENDIX

$P_{x, y\text{-}z}$, where P=p-value and x=within group, y-z=test between variable y and z. Fe=Female, Ma=Male, Dr=Driver, '35-44'=35-44 years, etc.

TABLE A-I STATISTICS FOR FIG. 2.

	Drivers			Passengers front-seat		
	Females (n)	Males (n)	Total (n)	Females (n)	Males (n)	Total (n)
NI	108	495	603	34	15	49
ST	13	50	63	19	8	27
LT+MT	13	36	49	7	4	11

$P_{Fe,\ Dr\text{-}Pa}$=.799, $P_{Ma,\ Dr\text{-}Pa}$=.094, $P_{Dr,\ Fe\text{-}Ma}$=.182, $P_{Pa,\ Fe\text{-}Ma}$=.733

TABLE A-II STATISTICS FOR FIG. 3 (ONLY DRIVERS).

	Tow bar YES			Tow bar NO		
	Females (n)	Males (n)	Total (n)	Females (n)	Males (n)	Total (n)
NI	59	279	338	47	200	247
ST	8	25	33	5	24	29
LT+MT	9	26	35	4	13	17

$P_{Fe,\ Yes\text{-}No}$=.556, $P_{Ma,\ Yes\text{-}No}$=.490, $P_{Yes,\ Fe\text{-}Ma}$=.163, $P_{No,\ Fe\text{-}Ma}$=.750

TABLE A-III STATISTICS FOR FIG 4-5.

	Female				Male			
	LT+MT (n)	ST (n)	NI (n)	Tot (n)	LT+MT (n)	ST (n)	NI (n)	Tot (n)
16-24 years	0	0	6	6	2	2	11	15
25-34 years	0	2	16	18	4	7	71	82
35-44 years	9	5	33	47	6	18	136	160
45-54 years	2	4	29	35	12	12	122	146
55-64 years	2	2	15	19	7	10	108	125
>=65 years	0	0	2	2	4	1	24	29
Total	13	13	101	127	35	50	472	557

$P_{'35\text{-}44',\ Fe\text{-}Ma}$=.001, $P_{'45\text{-}54',\ Fe\text{-}Ma}$=1.000, $P_{Fe,\ '35\text{-}44'\text{-}\ '45\text{-}54'}$=.105, $P_{Ma,\ '25\text{-}34'\text{-}'35\text{-}44'}$=.737, $P_{Ma,\ '25\text{-}34'\text{-}'45\text{-}54'}$=.426, $P_{Ma,\ '25\text{-}34'\text{-}'55\text{-}64'}$=1.000, $P_{Ma,\ '25\text{-}34'\text{-}'>=65'}$=.203, $P_{Ma,\ '35\text{-}44'\text{-}\ '45\text{-}54'}$=.146, $P_{Ma,\ '35\text{-}44'\text{-}'55\text{-}64'}$=.570, $P_{Ma,\ '35\text{-}44'\text{-}'>=65'}$=.049, $P_{Ma,\ '45\text{-}54'\text{-}'55\text{-}64'}$=.478, $P_{Ma,\ '45\text{-}54'\text{-}'>=65'}$=.309, $P_{Ma,\ '55\text{-}64'\text{-}'>=65'}$=.221

TABLE A-IV STATISTICS FOR FIG. 6–7.

		Female				Male			
		LT+MT (n)	ST (n)	NI (n)	Tot (n)	LT+MT (n)	ST (n)	NI (n)	Tot (n)
50-59	kg	4	2	5	11	-	-	-	-
60-69	kg	6	5	9	20	0	2	4	6
70-79	kg	1	2	1	4	16	17	26	59
80-89	kg	0	4	1	5	10	15	33	58
90-99	kg	1	0	1	2	5	9	13	27
100-109	kg	-	-	-	-	4	3	5	12
>=110	kg	-	-	-	-	0	1	4	5
Total		12	13	17	42	35	47	85	167

$P_{Ma,\ '70\text{-}79'\text{-}'80\text{-}89'}$=.267, $P_{Ma,\ '70\text{-}79'\text{-}'90\text{-}99'}$=.433, $P_{Ma,\ '80\text{-}89'\text{-}'90\text{-}99'}$=1.000

TABLE A-V STATISTICS FOR FIG. 8–9.

		Female				Male			
		LT+MT (n)	ST (n)	NI (n)	Tot (n)	LT+MT(n)	ST (n)	NI (n)	Tot (n)
150-159	cm	1	0	2	3	0	0	1	1

160-169	cm	9	10	8	27	1	1	2	4
170-179	cm	2	3	8	13	17	18	37	72
180-189	cm	1	0	0	1	14	23	37	74
>=190	cm	-	-	-	-	3	5	8	16
Total		13	13	18	44	35	47	85	167

$P_{Ma,\ '170\text{-}179'\text{-}'180\text{-}189'}$=.547

TABLE A-VI STATISTICS FOR FIG. 10-11.

	Female				Male			
BMI	LT+MT	ST	NI	Tot (n)	LT+MT(n)	ST (n)	NI (n)	Tot (n)
<18.5	0	0	0	0	0	0	0	0
18.5- <25	9	7	14	30	16	24	40	80
25- <30	2	4	3	9	17	21	38	76
>=30	1	2	0	3	2	1	7	10
Total	12	13	17	42	35	46	85	166

$P_{Ma,\ '18.5\text{-}<25'\text{-}'25\text{-}<30'}$=1.000, $P_{'18.5\text{-}<25',\ Fe\text{-}Ma}$=.310

TABLE A-VII STATISTICS FOR FIG. 12.

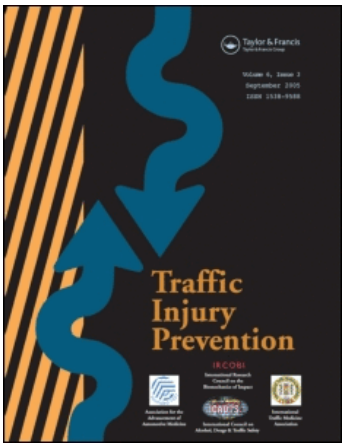
	Braking		Driving		Standing still	
	Br-Fe (n)	Br-Ma (n)	Dr-Fe (n)	Dr-Ma (n)	Sts-Fe (n)	Sts-Ma (n)
LT+MT	6	3	0	1	5	16
ST	5	13	0	1	6	23
NI	28	133	7	26	40	184
TOTAL	39	149	7	28	51	223

$P_{Br,\ Ma\text{-}Fe}$=.003, $P_{Sts,\ Fe\text{-}Ma}$=.549

TABLE A-VIII STATISTICS FOR FIG. 13-14

	Traffic light (n)	Queue (n)	Zebra crossing (n)	Give way (n)	Roundabout (n)	Give way, roundabout (n)	Stop sign (n)	Other (n)	Total (n)
LT+MT	4	4	3	0	3	1	0	2	17
ST	18	5	6	6	4	2	2	0	43
NI	42	46	39	40	17	10	11	23	228

$P_{Tr\ Li\text{-}Queue}$=1.000, $P_{Tr\ Li\text{-}Ze\ Cr}$=1.000, $P_{Tr\ Li\text{-}Gi\ Wa}$=.258, $P_{Tr\ Li\text{-}Round}$=.345, $P_{Queue\text{-}Ze\ Cr}$=1.000, $P_{Queue\text{-}Gi\ Wa}$=.244, $P_{Queue\text{-}Round}$=.380, $P_{Ze\ Cr\text{-}Gi\ Wa}$=.496, $P_{Ze\ Cr\text{-}Round}$=.319, $P_{Gi\ Wa\text{-}Round}$=.039



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The Anatomy and Biomechanics of Acute and Chronic Whiplash Injury

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The Anatomy and Biomechanics of Acute and Chronic Whiplash Injury

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Whiplash injury is the most common motor vehicle injury, yet it is also one of the most poorly understood. Here we examine the evidence supporting an organic basis for acute and chronic whiplash injuries and review the anatomical sites within the neck that are potentially injured during these collisions. For each proposed anatomical site—facet joints, spinal ligaments, intervertebral discs, vertebral arteries, dorsal root ganglia, and neck muscles—we present the clinical evidence supporting that injury site, its relevant anatomy, the mechanism of and tolerance to injury, and the future research needed to determine whether that site is responsible for some whiplash injuries. This article serves as a snapshot of the current state of whiplash biomechanics research and provides a roadmap for future research to better understand and ultimately prevent whiplash injuries.

Keywords Whiplash injury; Biomechanics; Neck; Injury mechanisms; Tolerance; Acute and chronic injury

INTRODUCTION

Neck sprains and strains—commonly known as whiplash injuries—are the most common motor vehicle injuries treated in U.S. hospital emergency departments (Quinlan et al. 2004). Incidence rates for whiplash injury range from 28 to 834 per 100,000 each year (Cassidy et al. 2000; Ostremski et al. 1989), and data stratified on gender and age show that females aged 20 to 24 have the highest incidence (~965 per 100,000 annually; Quinlan et al. 2004). Chronicity rates for whiplash patients also vary widely. In one study, 66 percent of subjects had residual neck pain after two years (Norris and Watt 1983), whereas in another study only 6 percent of subjects had residual neck pain after one month (Schrader et al. 1996). Such wide-ranging incidence and chronicity rates may stem from differing sample sizes, sampling methods, and injury definitions, but despite these differences, acute and chronic whiplash injuries are by a wide margin the most frequent automobile-related injury (Viano 2003). It also remains one of the most poorly understood automotive injuries.

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Clinically, whiplash patients present with neck, shoulder, or back pain; headaches; dizziness; paresthesias; vertigo; or cognitive/psychological symptoms (Evans 1992; Radanov et al. 1995; Sterner and Gerdle 2004). The source of the initial symptoms is often uncertain (Binder 2007), but it is generally presumed these initial symptoms have an organic basis. Multiple anatomical sites in the neck have been postulated for this initial injury, including the facet joints, spinal ligaments, intervertebral discs, vertebral arteries, dorsal root ganglia, and neck muscles (Figure 1, Table I). Some chronic pain also appears to be organic in nature (Lord et al. 1996a; Sterling 2006), although late whiplash syndrome is viewed by some not as a chronic injury but rather as a self-perpetuating cycle of maladaptive behaviors possibly initiated by an acute organic lesion (Ferrari and Schrader 2001).

Despite these disparate views regarding their origin, some symptoms of whiplash injury likely have organic bases that are related in some way to the forces transmitted through the neck and the strains experienced by tissues in the neck during a collision exposure. Indirect evidence supporting this premise is the 31 to 75 percent reduction in whiplash injuries reported for collisions in vehicles with new anti-whiplash seats designed to reduce these forces (Farmer et al. 2003; Jakobsson and Norin 2004; Viano and Olsen 2001). If there was no underlying injury caused by the collision exposure, then these new seats

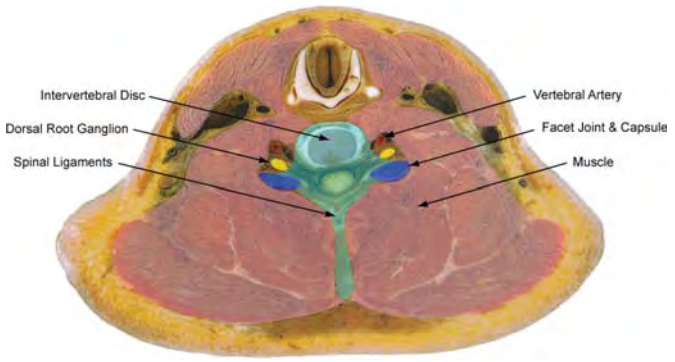

Figure 1 Cross section of the neck showing the anatomical arrangement of the proposed sites of whiplash injury. The shaded areas show muscle (pink), spinal ligament (aqua), facet joints (blue), dorsal root ganglia (yellow), vertebral arteries (red), and intervertebral disc (grey). (Adapted from Rohen and Yokochi 1993.)

would presumably have little or no effect on the rate of injury. Moreover, some whiplash injuries likely do not resolve for organic reasons rather than psychosocial ones. This latter proposition is supported by the delayed recovery and higher chronicity rates for patients with more severe initial symptoms (Scholten-Peeters et al. 2003; Suissa et al. 2001; Williams et al. 2007). What remains unclear, however, is whether chronic pain originates from the acutely injured tissue or whether other physiologic processes account for the persistence of pain.

Here we review the evidence supporting an organic basis for acute and chronic whiplash injuries. For each proposed anatomical site of whiplash injury—facet joints, spinal ligaments, intervertebral discs, vertebral arteries, dorsal root ganglia, and neck muscles—we present the clinical evidence supporting that injury, the relevant anatomy, the mechanism of and tolerance to injury, and the future research needed to definitively determine whether that site is responsible for some whiplash injuries.

FACET JOINT AND CAPSULAR LIGAMENT

Clinical Evidence of Injury

The cervical facet joints are the most common source of neck pain (Aprill and Bogduk 1992; Barnsley et al. 1994). Medial branch blocks and provocative testing have also implicated the facet joint in neck pain, particularly in chronic whiplash patients (Barnsley et al. 1993; Bogduk and Marsland 1988). This strong clinical evidence of facet-mediated neck pain has led to the development of diagnostic tests (e.g., facet blocks) and treatment procedures (e.g., radiofrequency neurotomies) that can reduce or eliminate pain for a period of time (Lord et al. 1996b).

Relevant Anatomy

There are two facet joints between each pair of cervical vertebra from C2 to C7. The facet joint is a synovial joint enclosed by a thin, loose ligament known as the facet capsule. A synovial fold on the inner capsule extends between the margins of the articulating bony surfaces. The facet capsule itself lacks the stiffness to alter the intervertebral kinematics and instead follows the motions of its surrounding bony vertebrae (Winkelstein et al. 2000).

Cervical facet joints are innervated by the medial branches of the dorsal primary ramus from the two levels surrounding each joint (Lang 1993). Several histologic and anatomic studies have identified mechanoreceptors and unmyelinated nociceptors in the cervical facet joint (Giles and Harvey 1987; Inami et al. 2001; Kallakuri et al. 2004; McLain 1994; Ohtori et al. 2003). Though the size of the receptive fields of these pain fibers remains unknown, it has been proposed that each fiber innervates an area large enough to collectively cover the entire joint (Cavanaugh 2000). The facet capsule also contains Aδ- and C-fibers, both of which transmit nociceptive singals; i.e., pain (Cavanaugh 2000; Cavanaugh et al. 1989; Giles and Harvey 1987; Inami et al. 2001; Kallakuri et al. 2004; McLain 1994; Ohtori et al. 2003). Nociceptors reactive for substance P and calcitonin gene-related peptide have also been identified in the cervical facet capsules (Inami et al. 2001; Kallakuri et al. 2004; McLain 1994; Ohtori et al. 2003). Both of these neuropeptides are neurotransmitters and nociceptive neuromodulators (Ma and Eisenach 2003; Munglani et al. 1996). Thus, the cervical facet joints have the necessary anatomical features to initiate and potentially modulate more widespread neck pain.

Injury Mechanism and Tolerance

The motion of the facet joint articular processes and the capsule during whiplash-like impacts have been characterized in both human volunteers and cadaveric specimens (Cusick et al. 2001; Kaneoka et al. 1999; Pearson et al. 2004). Based on documented joint motion, two mechanisms of facet joint injury have been proposed: pinching of the synovial fold and excessive strain of the capsule. Ono et al. (1997) and Kaneoka et al. (1999) observed that the cervical vertebrae rotate about a higher instantaneous

Table I Summary of the potential location, type, and duration of the injuries sustained at each proposed anatomical site of whiplash injury

Anatomical site	Specific site or level	Type of injury	Possible duration of injury or related pain		
			<1 Month	1–6 Months	>6 Months
Facet joint	C2/3 to C7/T1	Synovial fold pinching; excess capsule strain	Yes	Yes	Yes
Ligaments	Occiput to T1	Excess strain	Yes	Yes	Yes
Vertebral artery	Occiput to C6	Excess strain/pinching	Yes	Yes	Yes
Nerve root	C3 to T1	Cell membrane dysfunction	Yes	Yes	Yes
Muscles	Multiple muscles, each with associated tendons and fascia	Excess strain while active	Yes	No, but may mediate other pain sources	No, but may mediate other pain sources

center during a whiplash exposure than during normal voluntary motion and proposed that this abnormal motion compresses the posterior facet surfaces together, pinching the synovial fold. Although the synovial folds are innervated by nociceptors (Inami et al. 2001), no further work attempting to isolate this potential mechanism of whiplash pain has been performed.

Excessive facet capsule strain during whiplash has been demonstrated by numerous groups (Luan et al. 2000; Pearson et al. 2004; Yang and King 2003; Yoganandan et al. 2002). Peak strains of 29 to 40 percent have been measured in the C6/C7 capsule of cadaveric specimens exposed to whiplash dynamics, whereas peak strains experienced during normal bending are only 6 ± 5 percent (Panjabi et al. 1998a; Pearson et al. 2004). Head-turned postures can double peak capsule strain during simulated whiplash loading (Siegmund et al. 2008b). Prior to the occurence of tissue failure, partial ruptures of the facet capsule have been observed in both tension and shear loading of this joint (Siegmund et al. 2001; Winkelstein et al. 2000). Further, the maximum capsule strains at partial rupture (35–65 percent) do not exceed those strains observed in some capsules during the simulated whiplash loading (Siegmund et al. 2001; Winkelstein et al. 2000). These data suggest that capsule elongation during whiplash is a potential mechanism of injury in some individuals.

More recently, in vivo animal models have related facet joint biomechanics to afferent activity and pain symptoms. In a goat model, afferents in the facet capsule are activated by tensile loading of the C5/C6 facet joint (Lu et al. 2005a, 2005b). Capsule strains of 10 ± 3 percent activated nociceptive afferents, whereas strains of 44 to 47 percent were sufficient to saturate the mechanoreceptors and nociceptors. Similar strains in the C6/C7 capsule of the rat during joint distraction also produce persistent pain symptoms (Dong et al. 2008; Lee et al. 2004; Lee et al. 2004; Quinn et al. 2007). More importantly, however, the intensity and duration of persistent pain in the rat depend upon the magnitude of strain in the capsule. A maximum principal strain of about 21 percent is associated with persistent sensitivity (Dong et al. 2008; Lee et al. 2004; Lee et al. 2004b). These strains are consistent with those detected in the human capsule during whiplash simulations (Pearson et al. 2004; Siegmund et al. 2001; Sundararajan et al. 2004; Winkelstein et al. 2000). For the same levels of joint distraction that produce pain, the fiber organization in the capsular ligament is also altered (Quinn et al. 2007), indicating that collagen in the capsule is being disorganized by the joint distraction, despite the absence of complete ligament failure (Figure 2).

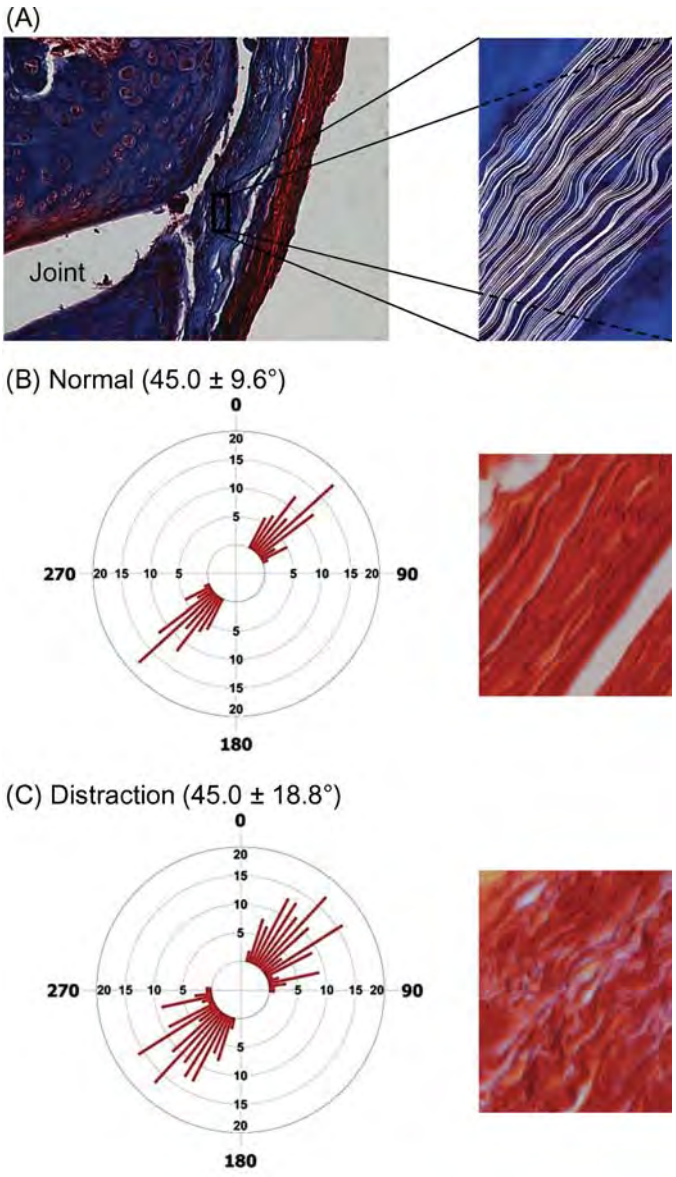

Figure 2 Facet capsule histology demonstrating collagen fiber organization in the rat facet capsular ligament. (A) Masson trichrome staining demonstrating a facet joint with its enclosing capsule (blue stain) and surrounding muscle (red). The inset shows a closeup of the facet capsule ligament with fibers—outlined in white—showing their typical undulation. (B), (C) Following a joint distraction sufficient to produce persistent pain symptoms, the collagen fiber organization (measured by angular deviation) is significantly larger. Shown here are representative histograms of angular deviation and sample tissue from (B) normal and (C) distracted ligaments.

Physiologic responses can contribute to pain in the absence of major mechanical failure. For instance, Lu et al. (2005a, 2005b) reported persistent after-discharges from afferents after joint loads were removed. At the cellular level, both neurons and other cells in the dorsal root ganglia demonstrate sensitive responses to painful and non-painful joint loading (Lee et al. 2008). Persistent increased expression of binding protein (BiP), a marker of cellular stress response (Dong et al. 2008), occurs predominantly in neurons of the dorsal root ganglia following painful facet joint loading similar to that which develops in whiplash. Inflammatory responses in the spinal cord are induced and sustained following painful joint loading and depend on the strain imposed on the capsule (Lee et al. 2004, 2008). These local and more widespread neuro-inflammatory cascades contribute to a variety of other chronic pain syndromes (DeLeo and Yezierski 2001). Their induction, persistence, and relationship to joint/capsule mechanics in painful whiplash loading supports the facet joint's involvement in whiplash pain.

Future Directions

Continued biomechanical research is needed to define how collagen injury during subfailure ligament loading initiates pain responses, their temporal response, and how such scenarios may be produced during whiplash. Moreover, continued research is needed to identify and define the specific physiologic pathways (electrophysiologic, immunologic, and otherwise) that are responsible for chronic pain following this joint's injury. Using this information, better diagnosis and treatment for facet-mediated, or at least facet-initiated, whiplash pain can be developed.

LIGAMENT AND DISC

Clinical Evidence of Injury

Magentic resonance and autopsy studies of whiplash patients have documented injuries to the neck ligaments and intervertebral discs in addition to the facet joints (Jonsson et al. 1991; Kaale et al. 2005a, 2005b; Krakenes and Kaale 2006; Pettersson et al. 1997). Whiplash-related symptoms may be due, in part, to injuries of cervical ligaments and discs and their embedded mechanoreceptive and nociceptive nerve endings. Ligament injuries may cause acute neck pain and lead to chronic spinal instability, and injured mechanoreceptors may corrupt normal sensory signals and could lead to abnormal muscle response patterns and decreased neck mobility and proprioception (Panjabi 2006).

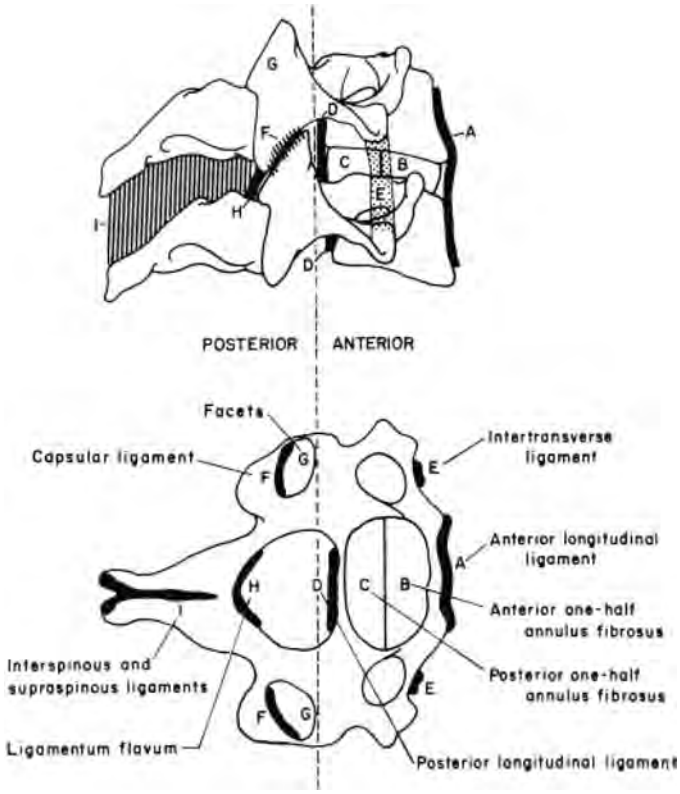

Figure 3 Ligaments of the middle and lower cervical spine (from White and Panjabi 1990).

Relevant Anatomy

The cervical vertebrae are joined by multiple ligaments. The main ligaments below the axis include the anterior and posterior longitudinal, capsular, interspinous, and supraspinous ligaments and the ligamentum flavum (Figure 3). The anterior and posterior longitudinal ligaments are thin sheets of tissue that span the anterior and posterior surfaces of the vertebral bodies, respectively, and blend with the underlying annular fibers. The capsular ligaments, as described earlier, encase the facet joints. The interspinous ligaments join adjacent spinous processes and are not present in all adults. When present, these ligaments are thin, weak tissues of high collagen content that blend posteriorly with the supraspinous ligament. The ligamentum flavum is the most elastic tissue in the human body—comprised of up to 80 percent elastin—and joins adjacent laminae bilaterally (Yahia et al. 1990). The intervertebral disc, located between adjacent vertebral bodies, consists of a central nucleus pulposus encased by annulus fibrosis fibers.

Ligaments of the upper cervical spine—occiput through the axis—have unique functional and structural anatomy. Alar and transverse ligaments play key roles in providing stability in this region due to the absence of intervertebral discs and the horizontal alignment of the facet joints (Dvorak et al. 1988). These ligaments have a high collagen and low elastin content, predisposing them to partial or complete rupture at low strains during high-speed elongation (Panjabi et al. 1998b).

Ligaments provide joint position sense during normal motion and combined with discs provide passive stability and absorb energy during high-speed trauma. The specific function of each cervical ligament and disc in resisting whiplash loading is dependent upon its specific anatomical location, orientation, geometry, and unique mechanical properties.

Injury Mechanism and Tolerance

Spinal ligaments and annular fibers encapsulating the discs can partially or completely rupture when stretched beyond their physiological limit. The whiplash-related response of the cervical ligaments and discs have been quantified for frontal, side, and rear impacts using a whole cadaveric cervical spine model with muscle force replication and a surrogate head (Figure 4A; Ivancic et al. 2005). During rear impacts with the head facing forward, dynamic strains in the anterior longitudinal ligament and annular fibers above physiological levels (Ivancic et al. 2004; Panjabi et al. 2004a) and increased joint laxity (Ito et al. 2004) were observed. The C5/C6 disc was found to be at highest risk of injury during both frontal and rear impacts (Ito et al. 2005). In addition to the C5/C6 disc, excessive strains were observed in superior discs, including C2/C3, during frontal impacts. The disc injuries occurred at lower impact accelerations during rear impacts compared to frontal impacts. During frontal impacts, the supraspinous ligament, interspinous ligament, and ligamentum flavum at C2/C3 through C7/T1 are at risk for injury due to excessive strain (Panjabi et al. 2004b). The T1 horizontal acceleration at which ligament and/or disc injuries were detected in those studies using pre- and post-impact flexibility tests was 5 *g*

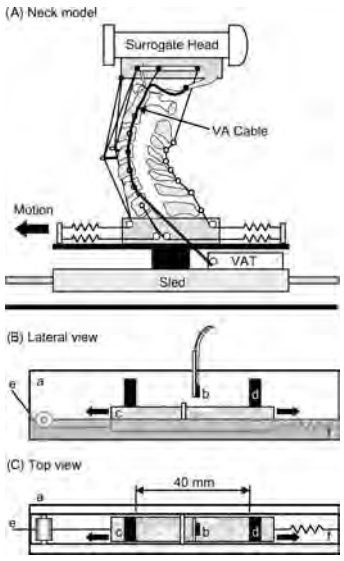

Figure 4 (A) Schematic of the biofidelic whole cervical spine model with surrogate head and muscle force replication used to simulate whiplash, showing the position of vertebral artery transducer (VAT) and vertebral artery (VA) cable passing through the foramen and attaching to the occiput. (B) Lateral and (C) top schematic views of the vertebral artery transducer: frame (a), Hall effect sensor (b), movable carriage (c) carrying two rare earth magnets (d), vertebral artery cable (e), and tension spring (f). (Adapted from Ivancic et al., 2006.)

for rear impacts, 6.5 *g* for side impacts, and 8 *g* for frontal impacts. For all impact configurations, the spinal levels at greatest risk of ligament and/or disc injury were C3/C4 through C7/T1.

Injuries to the alar and transverse ligaments of the upper cervical spine are reportedly more severe in individuals who have their head rotated at impact (Kaale et al. 2005a). These injuries have not been reproduced in cadaveric studies at impact accelerations (applied at T1) up to 8 *g* (Hartwig et al. 2004; Maak et al. 2006). This suggests that the upper cervical spine symptomatology reported by some whiplash patients may be due to impacts causing T1 accelerations in excess of 8 *g* or from some other anatomical structure.

Future Directions

Additional research is needed to further our understanding of ligament and disc injury mechanisms during whiplash and to investigate preventative mechanisms. Biomechanical studies are needed to correlate increased ligament and disc laxity with specific ligament injuries for each impact configuration. Future work is also needed to correlate the severity of ligament and disc injuries, in the form of biomechanical instability, with the onset of neck pain and, ultimately, to link specific ligament injuries to neck pain, or pain patterns, in whiplash patients. Biomechanical studies of simulated whiplash are needed to determine whether dynamic neck loads and high-speed ligament and disc strains are reduced by implementing specific injury prevention systems; e.g., active head restraint or energy-absorbing seat. These results may be correlated with those of epidemiological studies that investigate the effectiveness of injury prevention systems in reducing neck injury in real-life automobile collisions.

VERTEBRAL ARTERY

Clinical Evidence of Injury

Altered blood flow rates due to spasm and/or narrowing of vertebral arteries in whiplash patients have been associated with chronic symptoms of headache, blurred vision, tinnitus, dizziness, and vertigo (Reddy et al. 2002; Seric et al. 2000). Intimal tears of the vertebral artery are most common at the primary site of cervical axial rotation, the atlanto-axial joint (Barton and Margolis 1975; Chung and Han 2002; Davis and Zimmerman 1983; Pollanen et al. 1996; Sherman et al. 1981; Stahmer et al. 1997; Taneichi et al. 2005). Vertebral artery injury causing inadequate perfusion of the brainstem and surrounding tissues could explain some of the whiplash-related symptoms (e.g., headache, dizziness, and vertigo).

Relevant Anatomy

The vertebral arteries supply blood to the head, brain, and neck tissues. The vertebral arteries enter the spine at the C6 transverse processes bilaterally and run superiorly in the transverse foramen of each cervical vertebra. After exiting C1, the vertebral arteries travel along the C1 posterior arch and enter the foramen magnum of the skull. The vertebral artery is a viscoelastic structure: the adventitia is composed primarily of collagen fibers and the media consists of collagen as well as more substantial portions of smooth muscle and elastic fibers. It is encased in a fibrous tunnel and affixed to adjacent structures via a traneculated collagen network (Chopard et al. 1992).

Injury Mechanism and Tolerance

Coupled extension and axial rotation of the upper cervical spine has been hypothesized to cause vertebral artery injury (Barton and Margolis 1975; Chung and Han 2002; Davis and Zimmerman 1983; Sherman et al. 1981). Vertebral artery elongation causes a decrease in the vessel diameter due to Poisson's effect and could cause transient vascular compromise (Dobrin 1978). Alternatively, stretching or pinching of the vessel along a turn in its circuitous course is also possible (Barton and Margolis 1975). These mechanisms can also precipitate tearing of the intimal layer of the vertebral artery (Chung and Han 2002).

Cadaveric neck models have demonstrated coupled extension and axial rotation during side and rear impacts with the head turned but not during frontal or rear impacts with the head facing forward (Carlson et al. 2007; Ivancic et al. 2006). In those studies, average vertebral artery elongation was measured between the occiput and C6 vertebra using a custom transducer mounted in a cadaveric neck (Figure 4B). Peak vertebral artery elongation of 30.5 mm during head-turned rear impacts and 17.4 mm during side impacts significantly exceeded physiological elongation limits. Moreover, peak elongation occurred early—about 85 ms following the onset of T1 acceleration—with elongation rates reaching 1340 mm/s during head-turned rear impacts and 610 mm/s during side impacts. The magnitude, rate, and timing of vertebral artery elongation are thus sufficient to potentially cause vertebral artery injury.

Future Directions

Further biomechanical research is needed to determine the strain distribution throughout the vertebral artery during physiological movements and whiplash-related loading rates from different initial neck postures and in various impact directions.

DORSAL ROOT GANGLION AND DORSAL ROOT

Clinical Evidence of Injury

The dorsal root ganglion contains the cell bodies of most peripheral sensory nerves at each spinal level. Direct injury to cell bodies within the dorsal root ganglion could thus explain many of the typical whiplash symptoms (e.g., neck pain, cervicogenic headache, vertigo, vision disturbance, and neurological symptoms in the upper extremities). Generalized hypersensitivity to pressure acutely and chronically and decreased thermal pain thresholds in the skin over the cervical spine can be explained by impaired local sensory processing (Greening et al. 2005; Kasch et al. 2001b; Scott et al. 2005; Sterling et al. 2003, 2006; Sterner et al. 2001). In addition, increased electrical activity in the spinal cord and widespread reductions in electrical and pressure thresholds after whiplash suggest altered central pain processing (Banic et al. 2004; Curatolo et al. 2001; Kasch et al. 2001a; Scott et al. 2005). Increased sensitivity to pain (hyperalgesia) and larger areas of referred pain are also reported for whiplash patients (Koelbaek Johansen et al. 1999). These studies documenting both local and referred pain after whiplash injury provide clinical evidence for altered sensory transmission and pain pathways in the central nervous system.

Relevant Anatomy

The anterior and posterior rootlets coming off the spinal cord combine to form dorsal and ventral nerve roots, which make up the spinal nerves at each spinal level. The location, direction, and number of nerve rootlets vary at each cervical level. The dorsal and ventral roots come together in the region of the neural foramen and continue more distally into the periphery as the spinal nerve to innervate structures outside the spinal column. Posterior rootlets making up the dorsal root are the sensory (afferent) fibers, whereas the anterior rootlets making up the ventral root are the effector (efferent) fibers. Cell bodies of peripheral afferents are housed in the dorsal root ganglion, which has been shown to be particularly sensitive to loading—even slight compression of normal dorsal root ganglia can produce sustained electrical activity and pain (Howe et al. 1977). Unlike peripheral nerves, the nerve roots themselves are not enclosed by a thick epineurial sheath, and thus they lack the mechanical strength of their peripheral counterparts, potentially exposing nerve roots to increased risk of injury when loaded.

Injury Mechanism and Tolerance

Movements of the cervical spine in flexion, extension, and lateral bending cause the volume of the spinal canal to change. During normal voluntary neck motions, blood volumes in the internal and external vertebral venous plexa can easily move to compensate for these volume changes. During rapid whiplash-induced motions, however, resistance to blood flow and the inertia of the fluid mass itself can generate transient pressure gradients between the inside and outside of the spinal canal (Aldman 1986). These pressure gradients can directly load the spinal ganglia and nerve roots, potentially leading to whiplash-related symptoms.

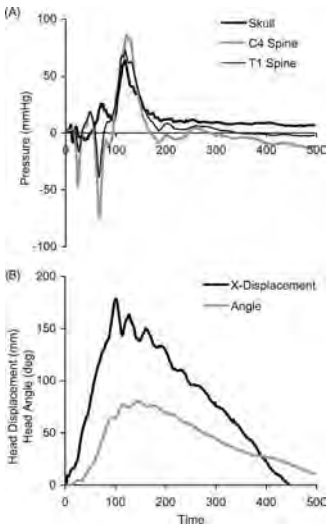

Figure 5 Pressure and displacement during a whiplash extension experiment using a pull-force on the porcine head-plate of 600 N. Graphs show (A) the pressure in the CNS at the skull, C4 and T1 vertebral levels, and (B) the angular and linear X-displacement of the head center of mass. (Adapted from Svensson et al., 2000.)

Whiplash experiments carried out on anesthetized pigs in extension, flexion, and lateral bending revealed a transient pressure drop inside the spinal canal during rapid motion in all directions (Figure 5; Svensson et al. 2000). Follow-up histology showed leakage of the plasma membrane of spinal ganglia nerve cells consistent with cellular injury (Örtengren et al. 1996). Eichberger et al. (2000) reported similar pressure recordings in cadavers exposed to whiplash and Schmitt et al. (2003) have since recreated the pressure pattern in a computational fluid dynamics model of the human cervical spine. These experimental findings are supported by an autopsy study of individuals who had sustained severe inertial neck loading (Taylor et al. 1998). Interstitial hemorrhage in the cervical dorsal root ganglia was observed in those autopsies despite an absence of injury to other structures surrounding the ganglia.

The relationship between the head-neck motion and the pressure magnitude in the spinal canal is quantified by the neck injury criterion (NIC; Boström et al. 2000). NIC is related to the relative horizontal acceleration and velocity of the head with respect to the torso, and a low NIC equates to a low risk of long-term neck injury (Krafft et al. 2003). Because many other loads and strains within the neck tissues also vary with NIC, this relationship between NIC and long-term neck injuries is not proof that dorsal root ganglion injuries explain all long-term whiplash injuries.

Deformation of the nerve roots themselves is another potential mechanism for producing persistent neck pain. The neural

foramina change shape and decrease their diameter during extreme neck motions (Carter et al. 2000; Krivickas and Wilbourn 2000; Yoo et al. 1992). This can compress the nerve root within the intervertebral foramen during whiplash motions. Nuckley et al. (2004) reported a 20 percent decrease in area for the C4-C7 intervertebral foramina of cadaveric cervical spines in extension. The intervertebral foramen at C5/C6 narrowed by as much as 1.8 mm during simulated rear impacts of a cadaveric head-neck model using horizontal T1 accelerations up to 8 *g* (Panjabi et al. 2006; Tominaga et al. 2006). This dynamic narrowing of the foramen during whiplash may compress the nerve roots and ganglia in the lower cervical spine, particularly in individuals with congenitally narrow foramen or those with osteophytes.

Transient loads on the cervical dorsal nerve roots have produced significantly elevated pain symptoms in a rat model (Hubbard and Winkelstein 2005; Hubbard et al. 2008; Rothman et al. 2005). Wallerian degeneration, disrupted axonal transport, and altered neuronal responses in the dorsal root ganglion are also produced (Hubbard and Winkelstein 2008). These data further support direct and indirect relationships between tissue loading, neuronal function, and altered physiology locally, in the dorsal root ganglia and throughout the nervous system for painful loading conditions.

Future Directions

Refined finite element and fluid dynamics models of the human head and neck may lead to better understanding of the flow and pressure phenomena that appear to result in ganglion dysfunction. This improved understanding would enable the development of more accurate injury criteria and tolerance limits for ganglion injury and would guide the development of improved crash dummies and performance requirements for injury protection systems in vehicles. Additional work is also needed to establish the link between the observed pressure transients and the generation and time course of ganglion dysfunction. The influence of nerve cell membrane dysfunction on nerve function and pain sensitization also needs to be investigated following experimentally induced ganglion injury.

MUSCLE

Clinical Evidence of Injury

Muscle or myofascial pain is a common symptom reported by whiplash patients (Evans 1992), although evidence of direct injury to muscle remains inconclusive. Injury-related muscle soreness is associated with a rise in serum creatine kinase detected at 3 to 24 h after high-intensity exercise and may persist for up to 9 days (Evans et al. 1986). In some whiplash patients, elevated serum creatine kinase has been observed 24 h after injury but not 48 h after injury, despite neck pain extending beyond 3 months (Scott and Sanderson 2002). Although this work suggests that direct muscle injury may not be responsible for chronic whiplash pain, muscles may nevertheless play an indirect role in modulating pain caused by injuries to other structures.

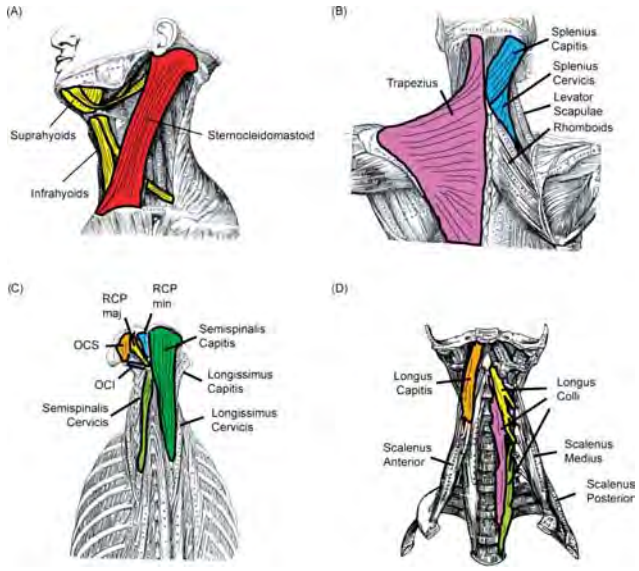

Figure 6 Neck muscle anatomy. (A) Lateral view of superficial muscles. (B) Posterior view of superficial muscles. (C) Posterior view of deeper muscles. (D) Anterior view of deep muscles. (Adapted from Gray, 1977.) (Figure appears in color online).

Relevant Anatomy

Muscles comprise the majority of the neck's volume (Figure 1). The superficial muscles, such as sternocleidomastoid or trapezius (Figures 6A and 6B), are often implicated in the pain and tenderness associated with whiplash injury. These superficial muscles attach to the skull, shoulder girdle, and ligamentum nuchae but do not generally attach directly to the cervical vertebrae. Deeper muscles, such as splenius, semispinalis, longissimus, scalenes, and longus, attach on multiple cervical vertebrae (Figures 6B, 6C, 6D). The deepest neck muscles, the multifidus muscles, insert directly on the facet capsule of cervical vertebrae (Figure 7) and may be relevant to injury of the capsular ligaments (Anderson et al. 2005; Winkelstein et al. 2001). Most neck muscles have complex architecture, with extensive internal tendon (Kamibayashi and Richmond 1998) and a high density of muscle spindles (Boyd-Clark et al. 2002). Although this has not been explored, the presence and arrangement of the internal tendon may be related to musculotendinous pain.

Injury Mechanism and Tolerance

The direct mechanism of neck muscle injury occurs from eccentric contractions; i.e., imposed lengthening during active contraction. Computer simulations using experimental kinematics of human subjects exposured to rear-end collisions have shown that both anterior and posterior neck muscles experience active lengthening during rear impacts (Brault et al. 2000; Vasavada et al. 2007). The anteriorly located sternocleidomastoid is active and lengthened during the retraction phase of whiplash, whereas posterior muscles are active and lengthened during the rebound phase. For simulated impacts with a speed change of 8 km/h, peak muscle fascicle strains averaged about 7 percent (max. 15%) in the sternocleidomastoid and 21 percent (max. 50%) in the posterior muscles such as semispinalis capitis. These

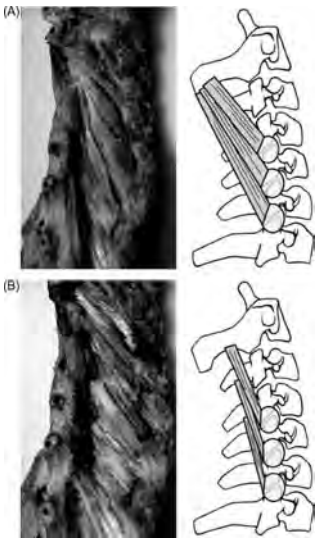

Figure 7 Anatomy of (A) superficial and (B) deep layers of the cervical multifidus muscles, depicting attachments on the facet capsules. (Adapted from Anderson et al., 2005.)

strains exceeded those shown to cause muscle injury (5–20%) in laboratory studies (Macpherson et al. 1996; McCully and Faulkner 1985). Thus, acute neck muscle injury may occur during rear-end impacts.

Interactions with Other Anatomical Sites

Neck muscles potentially interact with other anatomical sites of whiplash injury in at least three ways: (1) neck muscles attach directly to the facet capsule, which has been implicated in chronic pain following whiplash; (2) neck muscle activation indirectly affects the loads and strains in other anatomical structures; and (3) altered neuromuscular control may contribute to chronic pain via elevated and inappropriate muscle activation.

The pathomechanical evidence for facet capsular ligament involvement in whiplash injury and chronic neck pain has been outlined earlier in this article. Direct attachment of the multifidus muscles to the capsular ligament (Anderson et al. 2005; Winkelstein et al. 2001), combined with early activation of these muscles in some subjects during a rear-end collision may exacerbate peak strain in the capsular ligaments (Siegmund et al. 2008a).

Neck muscle activation also affects spinal tissue loads by increasing intervertebral compression and altering intervertebral kinematics. Because neck muscles are oriented primarily vertically, their activation produces axial compression of the cervical spine, increasing loads on the intervertebral disc and facet joints. Reflex muscle activation also affects the kinematic response of the head and neck. In subjects exposed to a series of identical perturbations, habituation of the muscle response amplitude by about 50 percent was accompanied by 10 to 30 percent changes in peak head kinematics (Siegmund et al. 2003). By altering head and neck kinematics, load and strain thresholds for injury may be exceeded in other structures such as ligaments, discs, and facet joints.

Finally, the interaction between muscles and the nervous system—i.e., via neuromuscular control—may be related to chronic pain. Patients with chronic pain demonstrate altered neuromuscular patterns (Falla et al. 2004; Nederhand et al. 2002), but it is not known whether the observed muscle activities are a physiological deficit in motor control or a protective strategy to avoid pain. A further complication is that different types of adaptive responses have been observed in different populations of whiplash patients (Nederhand et al. 2000, 2003). An inability to relax after exercise and excessive coactivation are associated with cervical pain (Elert et al. 1992; Nederhand et al. 2000; Westgaard et al. 1993), and relaxing selected neck muscles with botulinum toxin improves range of motion and reduces pain in these patients (Freund and Schwartz 2002). This suggests that pain and increased muscle activity may cyclically reinforce one another (Johansson and Sojka 1991). Contrasting evidence supports a pain adaptation model in which nociceptive interneurons inhibit the activity of painful muscles or those in the vicinity of pain sources (Lund et al. 1991). Nederhand et al. (2003) found that whiplash patients had a normal ability to relax the trapezius following exercise, but during exercise those with the highest disability levels had the lowest muscle activity. It remains unclear, however, whether muscle dysfunction is a cause (leading to damage of other anatomical structures) or effect (due to disuse or pain avoidance) of pain or merely an associated correlation.

Future Directions

Future research is needed to explore the role of neck muscles in the mechanism of acute whiplash injury, especially the interactions with other neck structures. Specifically, the effect of multifidus activity on capsular ligament mechanics and nociceptive physiologic responses needs to be studied to determine the relevant magnitude of loads from muscle forces on the ligament. Ideally, this type of research should be conducted in vivo, where muscles can be stimulated and ligament mechanical parameters measured. Research is also needed to explore how altered neuromuscular control relates to chronic pain. Specifically, studies are needed to analyze deep muscle activity in patients with chronic neck pain due to whiplash injury. In addition, validated mathematical models may be used to assess the effect of abnormal muscle activation on the loads in other anatomical structures.

SUMMARY

This review provides a brief summary of the anatomical structures being investigated by many groups to potentially explain whiplash injury. Each of the tissues described is strained during a whiplash exposure and thus could be injured if the crash-induced strain exceeds that tissue's tolerance. For each of the tissues summarized here, continued research is needed to better understand the biomechanical and physiological link between crash-induced loading and acute and chronic whiplash-related pain. A better understanding of each potentially injured tissue will help improve the diagnosis and treatment of whiplash injuries. Elimination or reduction of tissue strains through improved vehicle, seat, and head restraint design will help reduce the frequency of whiplash injury.

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Whiplash Mechanics in Low Speed Rear-End Automobile Collisions

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ABSTRACT. Objectives: To develop a biomechanical model that can help determine the exposure to potential physical injury based on the rebound flexion moment [acceleration load] of the head and neck.

Methods: By review of the biomechanical literature on rear-end collisions, to determine the impact force and the resulting acceleration to which the victim[s] had been exposed, and to determine the rebound flexion load acting on the cervical spine. To compare the developed formulation of the flexion load to measures taken from a staged rear-end impact experiment. To make adjustments to the injury threshold based on the above formulation, based on age, gender, and head position.

Results: In target vehicle velocity changes of 4.9 mph [7.8 kph] with peak vehicle acceleration of 3.0 g, the peak linear head acceleration was predicted to be 5.9 g, with peak rotational acceleration of 237 rad/sec^2. The resulting flexion moment about the occipital condyles was calculated to be about 20 ft-lbs [34 Nm], which was comparatively about

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46% of the tolerance for a normal adult male, 53% of the tolerance for an elderly male, 68% of the tolerance of a young female, and 101% of the tolerance for an elderly female. Non-normal population groups were not calculated.

Conclusions: Low velocity rear-end collisions should not likely expose a normal adult male or female to potential physical injury due to rebound flexion loading, but may expose an elderly female to injury.

KEYWORDS. Whiplash, biomechanics, flexion moment, collisions, rear-end impacts

INTRODUCTION

Whiplash injuries due to rear-end automobile accidents with minimal damage remain a significant and costly problem. Claims for these injuries account for between 68% and 85% of total automobile insurance costs (1). It has been estimated that between 35% and 42% of all medical claims for whiplash injury appear to be excessive (2). Controversy exists about the role that the potential for financial gain plays in the patient's perception of symptoms (3-8). The major impediment in determining the validity of the complaints of a victim of a low-damage rear-end vehicle collision remains the lack of an accepted objective medical technique for documenting the injury. Medical diagnoses therefore rely mainly on the patient's subjective descriptions of symptoms.

An approach which can aid in determining the exposure to injury is the use of biomechanical analysis. This method relies on specific physical evidence, the extent of structural damage to the automobiles involved in the collision, as a starting point to determining the impact force and the resulting acceleration to which the victim has been exposed. In this paper, a method is presented for estimating the flexion load acting on the cervical spine of the occupant in a rear-end collision. It is based on data from the biomechanics literature, combined with results of a staged rear-end impact experiment. The resulting flexion moment can be compared to the estimated threshold above which injuries might result. The injury threshold is adjusted for age, gender, and head position.

METHODS

The specific outcome measure on which the potential for injury is based in this analysis is the magnitude of the flexion moment acting on the cervical spine. It was chosen as a result of the following observations. Regardless of their complexity, the loads that the cervical spine is subjected to can be separated into six fundamental components. For the case of an upright seated, restrained individual in a seat with a head restraint, the resultant head-neck motion from a straight-on bumper to bumper collision takes place in the sagittal plane (9-17), eliminating consideration of axial rotation, lateral bending, and lateral shear as potential injury causing forces.

From a biomechanics perspective, four loads with probable injury causing potential act on the cervical spine during whiplash impacts. These are: 1. anterior shearing of the inferior vertebra during the phase when the torso rebounds forward out of the seat while the head is still falling backwards, 2. hyperextension of the cervical spine due to rearward displacement of the head, 3. compression due to head inertia as the torso ramps up the seat back, 4. hyperflexion in the rebound phase due to the head being unrestrained from its forward motion while the torso is restrained by the shoulder belt.

Matsushita et al. (10) used high speed radiography to study the motions of the cervical vertebrae of volunteers sitting in a standard head restraint equipped automobile seat, on a sled subjected to impacts up to 2 mph [5 kph]. They found no obvious signs of shearing or compressive displacement of the vertebrae on the radiographs, only extension and flexion. McConnell et al. (11) postulated that compressive loading might be a mechanism of cervical spine injury in whiplash. However, the stiffness coefficient of a cervical spine segment is approximately 200 N/mm in axial compression (17). Therefore a displacement as small as 1 mm vertically would require a load equivalent to about 5 g acting vertically on the head, which is well beyond the magnitude of the vertical force acting on the head in these low-damage impacts (11,15,18).

In experiments with normally seated occupants using appropriately positioned head restraints, several studies have shown that maximum neck extension ranged from 4° to about 60° (10-12,15,18) which is within reported ranges of normal spinal extension motions in vivo (17). Therefore unless the head restraint is poorly positioned, or the

occupant is significantly out of position, cervical spine extension should not exceed normal physiologic ranges in most accidents (19). This does contrast with the description given by Panjabi et al. (20) that the lower cervical spine may exceed its physiological range of extension, however, it should be appreciated that the model on which these observations were based was of an isolated cadaveric cervical spine with no spinal musculature, torso, seatback, or head-restraint. Ono et al. (21) using human volunteers whose cervical spine inter-segmental motions were tracked by high speed radiography, described a facet joint injury mechanism due to non-physiologic motions of individual vertebrae. These tests again were performed without head restraints. It is also interesting to note that although an injury mechanism was hypothesized from the measurements made, none of the subjects reported any post-test complaints.

This analysis focuses on the maximum flexion moment acting on the cervical spine as the head rebounds forward off the head restraint, which is a well documented motion of victims of rear-end impacts (10-12,14,15,18,19,22). The analysis was limited to impacts where a head restraint was available. Flexion is an unrestrained motion of the head caused by the torso motion being abruptly stopped by the shoulder belt upon rebound. Only tissue loading can stop the forward motion and downward rotation of the head, which has been accelerated off the head restraint.

In a study of victims of 237 real-world accidents, medical records indicate that the vast majority reported pain in the posterior aspect of the neck, along with the mid-back and shoulders, implying that these tissues were acting against the flexion loading of the neck (23). A prospective magnetic resonance imaging study of whiplash patients showed that 33% had disc herniation with medullary or dural impingement, without ligamentous injury (24). No anterior disc or anterior longitudinal ligament damage was observed. These types of injuries are likely to have resulted from flexion loading. Clemens and Burow (25) in a comprehensive study of the motions of cadaveric head-neck- and torso specimens during impacts, clearly identified flexion as the motion resulting in cervical spine injury, whether a head restraint was or was not present, because flexion is unrestrained and causes cervical spine tissue loading. Therefore in this analysis, flexion loading of the cervical spine is considered the most likely mechanism for tissue damage although it must be recognized that the analysis is limited to

upright normally seated occupants with appropriate head restraints where cervical extension would be limited.

Target Vehicle Acceleration

Three fundamental relations govern the mechanics of the impact, which is inelastic and includes tire forces: (26)

{1} Conservation of momentum:

$$m_tV_t + m_bV_b = m_bV_b' + M_tV_t' + [Fb + Ft]D_t$$

{2} Conservation of energy:

$$0.5m_bV_b^2 + 0.5m_tV_t^2 = 0.5m_bV_b'^2 + 0.5m_tV_t'^2 + C_b + C_t + B_b + B_t$$

{3} Restitution:

$$e = [V_b' - V_t']/[V_t - V_b]$$

Where:

m = mass of the vehicle
V = velocity of the vehicle at t = 0 [time of contact]
V' = velocity of the vehicle at t' [time of separation]
F = tire forces exerted due to braking
$D_t = t' - t$
C = work done in crushing vehicle components
B = work done due to braking
e = coefficient of restitution
subscript t = target or struck vehicle
subscript b = bullet or striking vehicle
superscrip t' = time of separation

In many cases, the actual crush energies are not known. This is because damage may not be observable for bumpers that do not show permanent deformation. In this case, neglecting the exact equation for energy conservation {2}, equations {1} and {3} can be combined to calculate a change in velocity of the target vehicle based on a worst case estimate of the approach velocity of the bullet vehicle:

$$\{4\} \quad V_t' = [1/[m_t + m_b]] * \{[m_b V_b [1 + e] - [F_b + F_t]\Delta t\}$$

The resulting acceleration of the struck vehicle has been shown to be approximately linear (10,27) and values for *e* are given in several sources (26,28-32) so the peak acceleration of the target vehicle is given by:

$$\{5\} \quad a_t = 2[V_t']/32.2*\Delta t$$

Head Kinematics

The following relationships between the peak acceleration of the vehicle and the linear and angular rotation of the head were derived from a review of a variety of published data, encompassing studies of rear impact kinematics, and plotted in Figures 1 and 2. This analysis assumes the worst case for torso motion, that the forward motion of the torso is arrested by the shoulder and lap belts, and is minimal. For head forward sagittal plane linear acceleration:

$$\{6\} \quad a_1 = 1.57 [a_t] + 1.52$$

Where:

a_1 = peak linear forward sagittal plane acceleration of the head, [g]

For head forward sagittal plane angular acceleration:

$$\{7\} \quad a_r = 99.7 [a_t] - 69.6$$

Where:

a_r = peak forward angular acceleration of the head in the sagittal plane, [rad/sec^2]

Moment Acting at the Occipital Condyles

The method described by Mertz and Patrick (22) was used to compute the bending moment of the head acting at the occipital condyles, assuming that the chin never contacted the chest. This was chosen for convenience since it is the location where a rigid mass, the head, acts at the end of a cantilever, the cervical spine. It should be pointed out

FIGURE 1. Relationship of vehicle peak acceleration and head forward linear peak acceleration taken from published reports of head kinematics [N = 124]. Data sources (5,9-16,18,19,21,22,25,27,34,37,40,41,43-45,49-58).

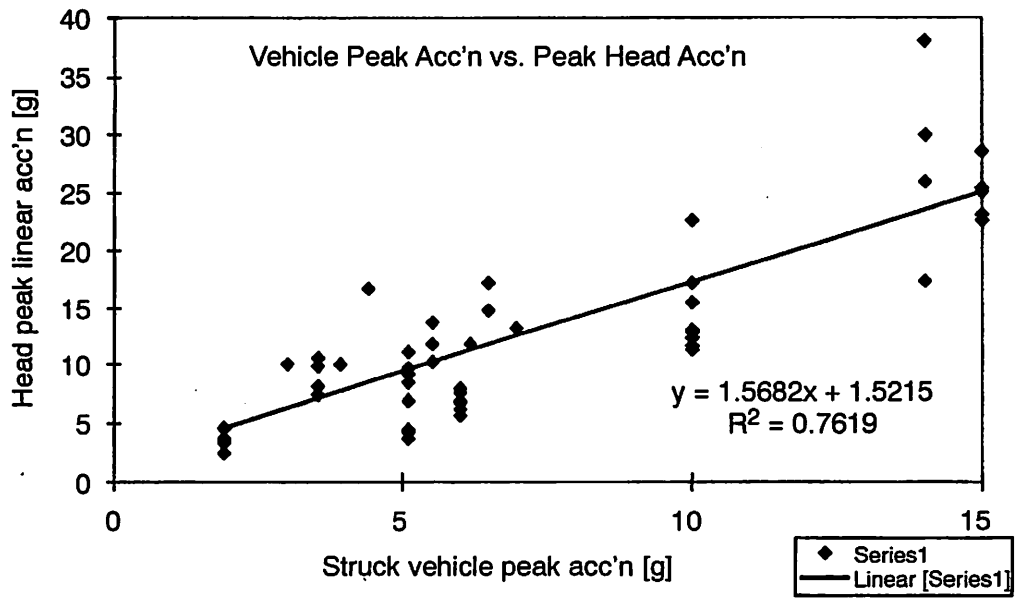
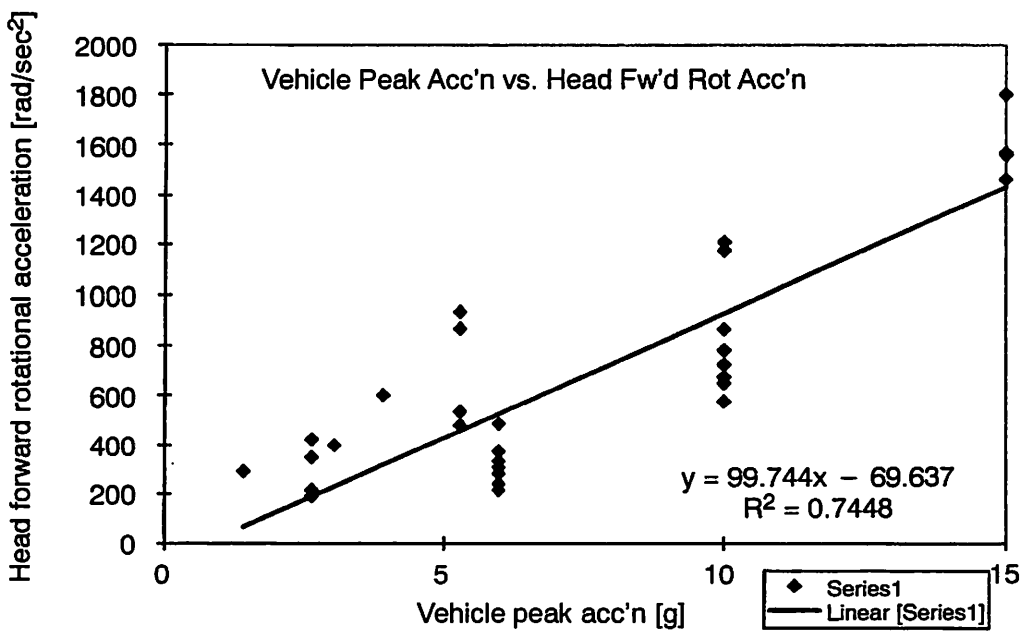


FIGURE 2. Relationship of vehicle peak acceleration and head forward angular peak acceleration taken from published reports of head kinematics [N = 33]. Data sources same as Figure 1.



that the C0-C1 joint acts as a hinge so only the bending moment due to the linear forward motion of the head actually causes bending of the cervical spine. Head rotary motion must be counteracted by the posterior muscles of the cervical spine. The resulting flexion moment acting on the cervical spine is given by:

$$\{8\} \qquad T = W * [1 + a_1] * r + I * a_r$$

Where:

T = resultant torque at the occipital condyles
W = weight of the head
r = distance from head cg to center of rotation of C0-C1 [atlanto-occipital] joint
I = mass moment of inertia of the head
Values for both w and I are readily available (33-35).

Estimating the Tolerance to Flexion Moments

Table 1 provides a summary of data from the literature, both from cadaveric studies and human volunteer testing, of the approximate flexion moments tolerated during testing. From these results, a mean value of about 44 ft-lbs [59.6 N-M] has been selected as a flexion moment tolerance for the normal human cervical spine. Table 2 provides estimated adjustment factors for spinal flexion loading based on the gender differences described by Siegmund et al. (15) and the differences in isometric cervical spine muscle strengths based on age and gender given by Faust et al. (36). The calculated flexion moment could therefore be compared to the estimated injury tolerance flexion moment, adjusted for age and gender.

Data Used in the Example

The following is provided as an illustration of the method. For the case of a collision of two modern vehicles, with energy absorbing bumpers which do not show obvious signs of deformation, the maximum approach velocity must be based on the threshold at which observable damage occurs (26). This data is readily available from sources such as Consumers Reports which uses a 5 mph [8 kph] pendulum impact test, or the Insurance Institute for Highway Safety

TABLE 1. Observed Thresholds for Flexion Moment Injuries in Cadaveric and Human Volunteer Studies of Rear End Impacts. Sled Acceleration in g, Neck Torque in ft-lbs

Sled Acc'n	Flexion Moment	Reference	Injuries
Cadaveric Studies			
20	49.0	Schmidt (14)	Minor to Disc
	140	Mertz (12)	Strain
	29.5	Gadd (51)	Strain
36	88.2	Clemens (25)	Disc Injuries
16.5	40.4	Clemens (25)	Disc Injuries
16	39.2	Kallieris (53)	20% Disc, C3-4
Human Volunteers			
5.0		Severey (19)	None
45.4	111.7	Stapp (37)	Minor
14	34.4	Patrick (54)	Minor
16.3	40.1	Armstrong (49)	Minor
14.8	36.4	Chandler (50)	Minor
9.9	24.4	Ewing (9,43)	Minor
21.2	52.2	Glenn (52)	Minor
15	36.9	Muzzy (34)	Minor
9.8		Wismans (58)	Minor
		West (57)	
3.6-6.8 mph		McConnell (11,18)	Minor
4.4-7.0		Szabo (27,38,55)	None
3.4 g		Seigmund (15)	Minor
mean 40.2, sd = 15.3, N = 45		Pincemaille (59)	[from Boxing]
5-6		Tencer (16)	Minor

TABLE 2. Adjustment Factors for Maximum Tolerance to Neck Flexion Based on Data of Isometric Muscle Strengths, After Foust et al. (36)

Age and Gender	Correction Factor
females 18-24	0.66
females 35-44	0.61
females 62-74	0.45
males 18-24	1.00
males 35-44	1.10
males 62-74	0.86

which uses an equivalent barrier impact. The differences in reported damage threshold velocities should be appreciated, and damage threshold data from different sources should be carefully assessed.

In this example, the damage threshold speed change of the bullet vehicle is assumed to be about 6 mph [9.7 kph]. Recognizing that as the vehicles impact they assume approximately the same speed, and with e = 0.3, the approximate approach speed [V_b] of the bullet vehicle for its assumed damage threshold speed is about 9 mph [14.5 kph]. For this example, the vehicles are of approximately equal mass, taken in this example as 2,800 lbs loaded [1,273 kg], the bullet vehicle is undergoing heavy braking, the target vehicle is stationary, and the approximate impact duration is 150 msec (18,26,27,31,32).

RESULTS

For the conditions given above, and using equation {4}, the resulting speed change or change of velocity [delta V, or ΔV] of the target vehicle [V_t'] was calculated to be about 4.9 mph [7.8 kph] with a peak acceleration of 3.0 g. From equation {6}, the peak linear acceleration of the head was predicted to be 5.9 g, and peak rotational acceleration was 237 rad/sec^2. The resulting bending moment acting at the occipital condyles, from equation {8} was about 20 ft-lbs [34 Nm].

In order to put the estimated cervical spine bending moment estimated in this example into perspective, using Table 1 and Table 2, the flexion moment predicted would reach about 46% of the tolerance for a young to middle age male, 53% of the tolerance of an elderly male, 68% of the tolerance of a young female, and 101% of the tolerance for an elderly female.

DISCUSSION

The estimates for head acceleration derived here are in good agreement with observations of volunteer responses in several studies devoted to staged rear end impacts between automobiles (11,14-16, 18,22,37,38). In our recently completed study (16), we found the ratio of peak head acceleration to peak vehicle acceleration to be 1.23, which is somewhat lower than the value of 1.57 in equation {6}

derived from the data extracted from the studies listed, and probably reflects the relatively compliant head restraint of the seat used in our study.

Based on the computed head accelerations and neck moments generated by these types of impacts, it is clear why volunteers in staged impact studies report at most, only minor and transient neck injuries. The observation that the neck rebound flexion moment is, for most victims of these accidents, below the estimated tolerance for damage to ligaments, facets and discs, does not necessarily imply that no damage to tissues occurs. In fact, most victims of rear-end accidents in our risk factor analysis of actual accidents (23) described minimal or no symptoms at the time of impact but progressive onset of stiffness and soreness over a period of 24-48 hours. This is completely consistent with minor soft tissue strain injury, which would not be expected to be of long duration, in comparison to significant damage to ligaments or discs.

It should be appreciated that the method as described uses a worst case analysis with respect to determining the acceleration of the head and the resulting torque on the neck. The peak acceleration of the head CG chosen here really represents the forward acceleration of the head during contact with the head restraint. It was chosen because it is the most widely reported value in testing. However in a recently completed study of human volunteers (16) we found that there are separate peaks for the acceleration and deceleration of the head. The forward acceleration peak occurs between about 200 msec and 225 msec after impact, when the backward falling head hits the forward moving head restraint. The deceleration of the forward motion of the head results from the restraining actions of the tissues of the neck, and occurs over a longer period of time [from about 350 msec to 450 msec], so is therefore lower. In fact, we found this deceleration to be about half the peak forward acceleration of the head (16).

A number of factors affect occupant responses and tolerances to impact. Because of differences in size of the neck and the musculature surrounding it in different individuals, age and gender obviously have an effect on neck tolerance to flexion loading. Foust et al. (36) showed this in their study of the maximum isometric neck extensor and flexor torque in young [18-24 years old], middle aged [35-44], and elderly [62-74] male and female volunteers. Siegmund et al. (15) showed that there was a small but statistically significant difference in the response

of male and female volunteers to rear-end impacts. From this data, approximate adjustment factors for neck flexion moment tolerance due to age and gender were developed. This allowed comparisons with the well defined cervical spine flexion tolerances derived from younger adult males which comprise the largest volunteer impact test sample. These are given in Table 2.

The example analyzed here assumes a normal upright driving condition, however, there are several modifiers of the response of the occupant. Those that have been investigated are the effect of initial neck muscle tension, that is being prepared for the impact, the horizontal distance of the head forward of the head restraint [leaning forward], the vertical distance of the top of the head above the head-restraint, a rotated head position, and the type of seat. Regarding the role of neck muscles, Hendler et al. (5) showed that muscle testing could significantly enhance the subject's ability to withstand higher impacts, with tolerable peak accelerations over 21 g. Both Pope et al. (39) and Szabo et al. (38) demonstrated, using human volunteers, that there is sufficient time for the neck muscles to maximally contract in reaction to a low speed impact. Therefore, regardless of the state of preparedness of the occupant before impact, the muscles probably play some role in deceleration of the head in a low speed impact.

Head position at the time of impact has been investigated. When the horizontal distance of the head to the head restraint is increased, for example, if the occupant is leaning forward at the time of impact, the velocity of the head as it hits the head restraint is increased. This is because the torso impacts the seatback leaving the head to fall backward as the torso is thrust forward (12,40). Figure 3 gives an estimate of the effect of head position relative to a standard distance of about 4.25 in [10.8 cm] between the back of the head and the head restraint.

The effect of vertical distance of the head above the head restraint and head rotation has recently been investigated (16). In testing of adult males and females with a range of statures such that the distance of the tops of their heads were between 10 and 25 cm above the top of the head restraint, there was no correlation between head peak acceleration and vertical distance. Having the head rotated about 45 deg to the left prior to impact had no significant effect on head accelerations in the sagittal plane. There was added lateral acceleration due to the head moving from its initially rotated position to its straight forward position.

Seat stiffness is also known to affect the acceleration of the head (40,41) although a comparison of the data from a variety of studies using standard automotive seats, Tables 3 and 4 indicate that the effect of this variable for production seats, is not excessively large. The effect of the seat in occupant motion in rear-end collisions has been recognized for some time, since interaction of the body and the seat causes the motions resulting in whiplash. Severy et al. (42) made the following important observations with respect to seat design: 1. Head to head restraint horizontal distance should be kept to a minimum, at

FIGURE 3. Relative effect of back-of-head to head-restraint distance on peak forward linear head acceleration compared with a distance of 4.25 in [10.8 cm].

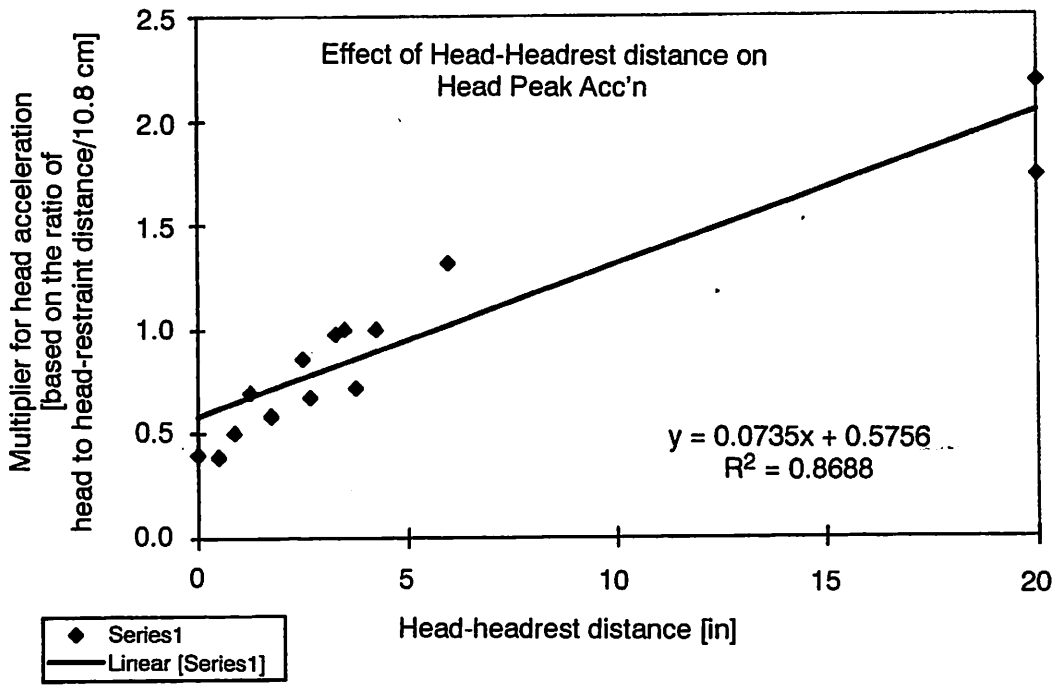


TABLE 3. A Comparison of Peak Head Accelerations of Occupants in Different Driving Conditions

Activity	Approximate head cg acceleration [g]	Reference
Impact in bumper car	1.0-1.4	Seigmund (32)
Backing up hard braking	0.44	Seigmund (26)
Backing up into barrier, 3 mph	5	Tender (16)

least below 3″. 2. Head rotation relative to the torso decreased as seat back height increased to 28″. 3. Increasing seat rigidity prevented the seat from bending backwards excessively which otherwise allows the occupant to ride up the seat back, resulting in the head falling back over it. Also increased seat rigidity and plastic deformation decreased the chances for forward rebound from the seat. 4. Contouring the upper third of the seat reduced the ramping of the occupant up the seat back. 5. The head restraint should be integral with the seat, so that excessive flexibility of the head restraint is minimized. Ewing et al. (9,43) using cadavers and human volunteers, Portnoy (44) using live baboons, and Berton (45), using dummies made similar observations. Melvin and McElhaney (46) stated that principles for rear-end protection include providing a seatback with minimal elastic energy storage potential, cushioning material with energy dissipation properties, a seat back contoured to minimize differential motions of the spinal column, and mechanical characteristics of the head restraint and lower portion of the seat matched to provide uniform motion of the occupant into the seat back and during rebound. Improvements in seat design have recently been described (47,48).

It should be recognized that the calculated tolerances as described above are quite conservative estimates based on two assumptions. First the acceleration exposure calculated is based on the damage threshold speed as the approach speed of the bullet vehicle. In many cases, with no visible damage to the vehicle, the actual approach speed may be much less. The threshold for vehicle damage is also specific to the type of vehicle involved in the collision. Secondly, there are two acceleration peaks of the head in the response. The first is when the head-restraint impacts the back of the head causing it to be accelerated forward off the seat. The second peak results from the slower deceleration of the head by the neck tissues, which halts its forward motion. The data used in this study was based on the first, higher acceleration peak, although several studies show that the second or deceleration peak is smaller (11,15,16,18).

Even though the forces applied in the impact may be too low to reach thresholds for injury, at least for the unrestrained forward flexion of the head and neck, the startle reflex, due to being surprised by the impact, may produce minor muscle symptoms. Szabo and Welcher (38) showed, in rear-end impacts, that the posterior muscles of the neck, particularly the trapezoids, may contract above their maximum

TABLE 4. A Comparison of Peak Head Accelerations of Occupants in Different Types of Vehicle Seats at Approximately the Same Vehicle Acceleration

Impact Acc'n [g]	Type of Vehicle	Peak Head Acc'n [g]	Reference
5.5	1981-2 Escort	11.9	Szabo (38)
6.1	1976 Volvo	14.6	Szabo (55)
5.3	1990 Honda Accord LX	9.0	Siegmund (32)
5.1	seat F1, no lumbar support frame	8.5	Svensson (41)
5.1	seat F2, no lumbar support frame, flexible seat back	9.1	Svensson (41)
5.1	seat F3, no lumbar support frame, flexible seat back	9.7	Svensson (41)
5.1	seat F4	6.8	Svensson (41)
5.1	seat F5, no lumbar support frame, flexible seat back	11.1	Svensson (41)

Note: The seat type was not identified in the study by Svensson (41).

voluntary levels in reaction to the head motion [mean = 83.5%, range 27.1% to 254.9%] presumably resulting in a contraction strain.

The method presented allows estimation of the maximum rebound flexion moment acting on the neck of the occupant in a typical rear end impact. If resisting the flexion rebound of the head is a primary mechanism of cervical spine tissue injury, then reduction of this flexion moment can reduce the load on the spine which presumably will reduce the potential for injury. An appropriately positioned head restraint, close to the back of the head and above its center of gravity, along with a seat that can absorb the impact of the torso and head against it, and with stiffness characteristics that reduce differential motions between the head and torso are possible modifications that may reduce cervical spine flexion loading in whiplash.

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CHANGE OF VELOCITY AND PULSE CHARACTERISTICS IN REAR IMPACTS: REAL WORLD AND VEHICLE TESTS DATA

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ABSTRACT

Impact severity in collisions that can cause soft tissue neck injuries are most commonly specified in terms of change of velocity. However, it has been shown from real-world collisions that mean acceleration influences the risk of these injuries. For a given change of velocity this means an increased risk for shorter duration of the crash pulse. Furthermore, dummy response in crash tests has shown to vary depending on the duration of the crash pulse for a given change of velocity. The range of duration for change of velocities suggested for sled tests that evaluate the protection of the seat from soft tissue neck injuries are still to be established. The aim of this study was to quantify the variation of duration of the crash pulse for vehicles impacted from the rear at change of velocities suggested in test methods that evaluate the protection from soft tissue neck injuries. Crash pulses from the same vehicle models from different generations in real-world collisions producing a similar change of velocity were also analysed.

The results from the crash tests show that similar changes of velocity can be generated with various durations of crash pulses for a given change of velocity in rear impacts. The results from real-world collisions showed that a similar change of velocity was generated with various durations and shapes of crash pulses for the same vehicle model.

INTRODUCTION

Rear impacts causing AIS 1 (AAAM 1990) neck injuries most frequently occur at delta-Vs (changes of velocity) below 30 km/h in the struck vehicle (Parkin et al., 1995, Hell et al., 1999, Temming and Zobel, 2000). Furthermore, it has been shown that mean acceleration (i.e. the duration of the crash pulse for a given delta-V) influences the risk of AIS 1 neck injuries (Krafft et al., 2002). It has also been shown that the shape of the crash pulse influences. risk of AIS 1 neck injuries in frontal impacts (Kullgren et al., 1999). Acceleration pulses from rear impacts shows that the same delta-V can cause a large variation in acceleration pulse shapes in the struck vehicle (Krafft, 1998, Zuby et al., 1999, Heitplatz et al., 2002). From real-world collisions it has been shown that the acceleration pulse also can vary in shape (i.e. duration of crash pulse, maximum magnitude of acceleration, onset rate etc) in impacts of similar delta-Vs (Krafft, 1998).

Dummy response in crash tests has been shown to vary depending not only on the delta-V but also on the duration of the crash pulse for a given delta-V (Linder et al., 2001a). The range of the duration of the crash pulse that corresponds to a specific delta-V in rear impacts has been shown to cover a wide range for vehicles impacted at the rear at a delta-V of up to 11 km/h (Linder et al., 2001b). The range of the duration of the crash pulse that corresponds to a specific delta-V in rear impacts that can cause AIS 1 neck injuries remains to be established. The range of the duration of the crash pulse for a specific delta-V is necessary to establish when designing impact severities for sled test methods that evaluate the safety performance of a seat in rear impacts, particularly in respect of AIS 1 neck injuries. Such test methods are at the moment under development Cappon et al. (2001), Muser et al. (2001), Langwieder and Hell (2002) and Linder (2002) and under discussion in groups like IIWPG (International Insurance Whiplash Prevention Group), EuroNCAP (European New Car Assessment Program), EEVC (European Enhanced Vehicle Safety Committee) Working group 12 and ISO (International Organization for Standardization) TC22/SC10/WG1. The delta-V suggested in sled test in these methods that represent the delta-V where the majority of rear impacts are reported is 15 or 16 km/h (Cappon et al., 2001, Muser et al., 2001 and Langwieder and Hell, 2002).

The first aim of this study was from laboratory crash tests to quantify the variety of mean acceleration monitored in different vehicles impacted in the same way. The second aim was to demonstrate the variety of the duration and shape of the crash pulse in the same vehicle model from real-world crashes producing similar delta-V.

MATERIALS AND METHODS

Laboratory Crash Tests

Sixteen vehicles (Table 1) were impacted at the rear either with a barrier or with a vehicle of the same make and model as the impacted vehicle. The barrier used in the OW test had a weight of 1000 kg (Figure 1). The barrier used in the CR tests had a weight of 1800 kg. The vehicles were impacted at the rear with 100 % overlap. The test where a vehicle was impacted by another vehicle (test OW3739, CR01001 and CR01002), the same make and model of vehicle was used as the bullet vehicle. The mass of the cars used were from 1010 kg - 1966 kg. The OW9999 vehicle was from 1983 series car (the actual vehicle was a used vehicle new in 1993 and with no structural corrosion) and the other vehicles were from the mid 1999.

Table 1.
The weight of the impacted vehicles and the impact velocity of the barrier or the impacting vehicle in the rear impacts.

Impact No.	Vehicle mass (kg)	Impact velocity (km/h)
OW9999	1190	18.3
OW3660	1450	30.0
OW3737	1965	52.4
OW3749	1445	36.9
OW3763	1347	35.7
OW3759	1493	32.1
OW3760	1493	43.8
OW3718	1010	40.0
OW3539	1384	24.9
OW3594	1405	35.2
OW3500	1339	18.5
CR98001	1450	24.0
CR98002	1800	24.0
CR98006	1750	24.0
CR01001	1439	32.0
CR01002	1461	32.0

Figure 1.

A rigid barrier impacting the rear of the vehicle.

One vehicle model was impacted both with a barrier and with another vehicle in order to compare the crash pulse from a rigid barrier to that generated by an impacting vehicle. The crash pulses from the laboratory tests were from previously performed tests at the Motor Insurance Repair Research Centre in the UK and at the Insurance Institute for Highway Safety in the US. The accelerometer was mounted at the base of the B-pillar on the left hand side in the vehicles in the OW tests. The vehicles in the OW tests were right-hand drive vehicles for the UK market. The vehicles in the CR tests were left-hand drive vehicles. The accelerometer was mounted on a steel bar pinned between the front door hinge-pillar and the b-pillar on the left hand side in the vehicles in the CR98001 and CR98002 tests. The accelerometer was mounted to the floor in the vehicle centreline just behind the front row of the seats in the vehicles in the CR98006 and the CR01 tests. The CR01 tests were performed with vehicles of the same make and model for the US and European market. These vehicles were structurally identical except from the bumper system. All vehicles were a conventional monocoque construction.

Real-World Rear Impacts

Since 1995, Folksam in Sweden have been equipping various new car models with one-dimensional crash-pulse recorders, mounted under the driver or passenger seat to record the crash pulse obtained during real-world impacts. The crash-pulse recorder is based on a spring mass system where the

movement of the mass is registered on photographic film. When a vehicle equipped with a crash recorder has been involved in a collision the crash pulse is analysed by Folksam and the outcome for the occupants in terms of injuries is analysed by Folksam. In this study, crash pulse from rear impacts with two generation of the same vehicle model, T1 and T2 from 1993 and 1998, were presented.

Data Acquisition and Analysis

The crash pulse measured as the acceleration signals of the vehicle were filtered in accordance with SAE CFC 60 and the velocity was calculated by integrating the acceleration. The duration of the crash pulse (T_p) and the delta-V were identified from the filtered acceleration curves and the velocity curves.

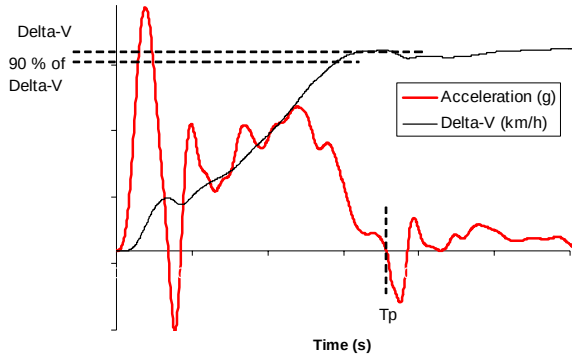

Figure 2.

Schematic drawing showing how the duration of the crash pulse T_p were identified from the graphs.

The T_p was defined as the time when the acceleration changed from positive to negative after 90 % of delta-V had occurred. Mean acceleration was calculated, defined as delta-V(at Tp)/Tp. For a given change of velocity a higher mean acceleration thus correspond to this a shorter duration of the crash pulse. For the graphic presentation of the crash pulses in this study the pulses were all adjusted so that the acceleration of 1 g occurred at time zero. Furthermore the crash pulses were filtered with CFC 36 (corresponding to a cut of frequency of 60 Hz) since oscillations in the crash pulses were found (Figure 3).

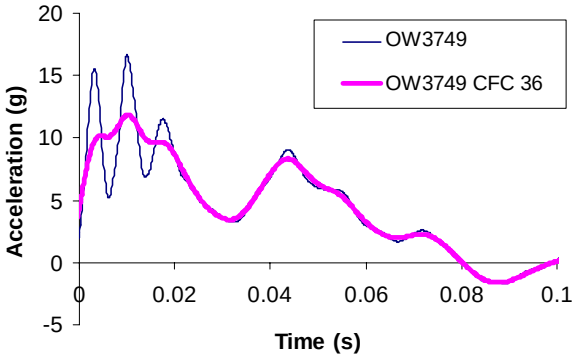

Figure 3.

Example of the oscillations filtered out by the CFC 36 filtering compared to the SAE standard filtering (CFC 60).

RESULTS

The results showed a considerable variation of the duration of the crash pulse for a similar delta-V both for different vehicles impacted the same way and for the same vehicle model impacted in various ways in real-world collisions. Furthermore, various pulse shapes were registered in the same vehicle from impacts which generated a similar delta-V.

Laboratory Crash Tests

The crash pulses from sixteen vehicles rear impacted with delta-Vs from 10.2 km/h to 19.4 km/h were examined. The duration of the crash pulse were between 65 ms and 130 ms. This resulted in mean accelerations between 3 g and 7.9 g (Figure 4 and 5 and Table 2).

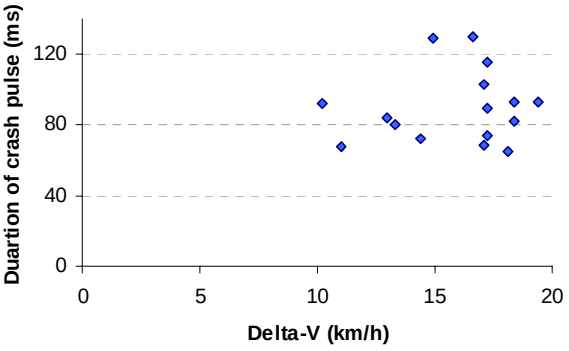

Figure 4.

The duration of the crash pulse versus delta-V from vehicles impacted at the rear with a rigid barrier or with another vehicle.

Table 2.
The delta-V, mean acceleration and duration of the crash pulse, T_p, from the vehicles impacted at the rear with a rigid barrier or another vehicle.

Impact No.	Delta-V (km/h)	T_p (ms)	a_{mean} (g)
OW9999	10.2	92	3.0
OW3660	17.1	69	7.0
OW3737	17.1	103	4.7
OW3749	18.4	82	6.4
OW3763	17.2	89	5.5
OW3759	18.4	93	5.6
OW3760	17.2	74	6.6
OW3718	18.1	65	7.9
OW3539	13.3	80	4.9
OW3594	13.0	84	4.4
OW3500	11.0	68	4.4
CR98001	16.6	130	3.6
CR98002	14.4	72	5.7
CR98006	14.9	129	3.3
CR01001	19.4	93	5.6
CR01002	17.2	116	4.1

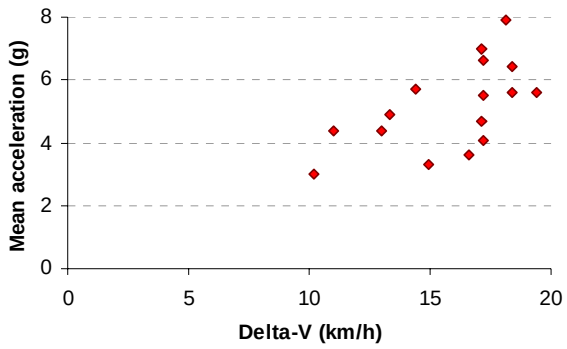

Figure 5.

The mean acceleration versus delta-V from vehicles impacted at the rear with a rigid barrier or with another vehicle.

The crash pulses from thirteen vehicles impacted with a rigid barrier (all OW tests except OW3759 and the CR98 tests) showed a range of mean acceleration from 3 g to 7.9 g. The crash pulses from the three vehicles impacted with another vehicle (OW3759 and CR01 tests) showed a range of mean acceleration from 4.1 g to 5.6 g.

The crash pulses recorded in the laboratorial tests are displayed in Figures 6-12.

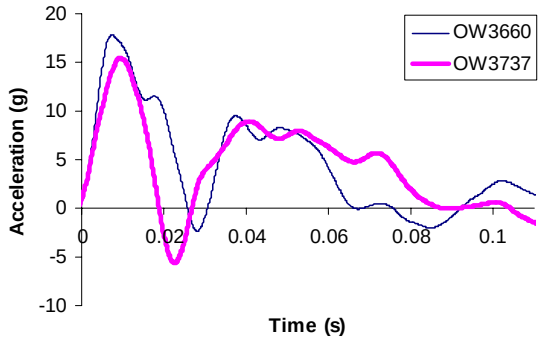

Figure 6.

The crash pulses from the OW3660 and OW3737, in tests at 100 % overlap with an impacting barrier generating a delta-V of 17.1 km/h.

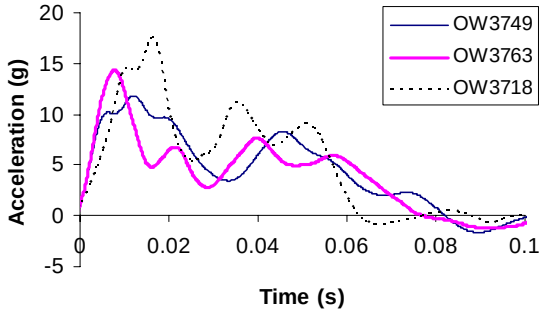

Figure 7.

The crash pulses from the OW3749, OW3763 and OW3718, in tests at 100 % overlap with an impacting barrier generating a delta-V of 17.2 km/h to 18.4 km/h.

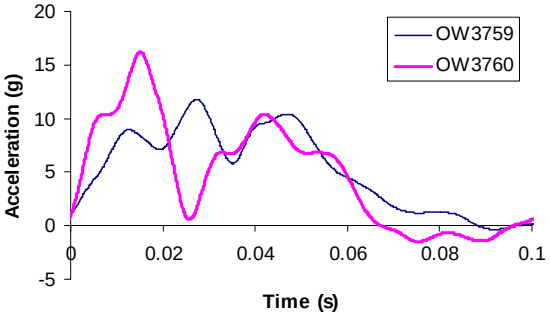

Figure 8.

The crash pulses from the OW3759 (car-to-car), and OW3760 (barrier to car), in tests at 100 % overlap with a delta-V of 17.2 km/h and 18.4 km/h.

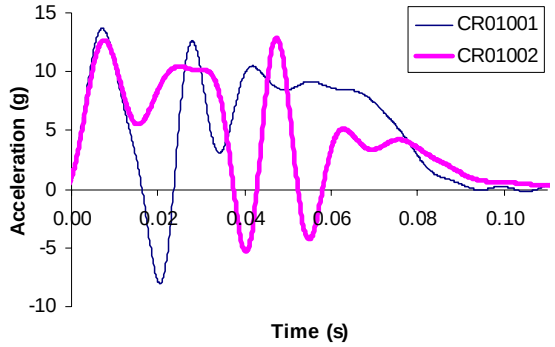

Figure 9.

The crash pulses from the car-to-car test with the same vehicle model for the US and European market, in tests at 100 % overlap with a delta-V of 17.2 km/h and 19.4 km/h.

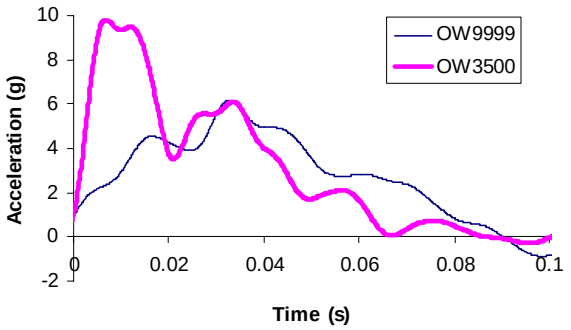

Figure 10.

The crash pulses from the OW3500 and OW9999, in tests at 100 % overlap with an impacting barrier generating a delta-V of 10.2 km/h to 11 km/h.

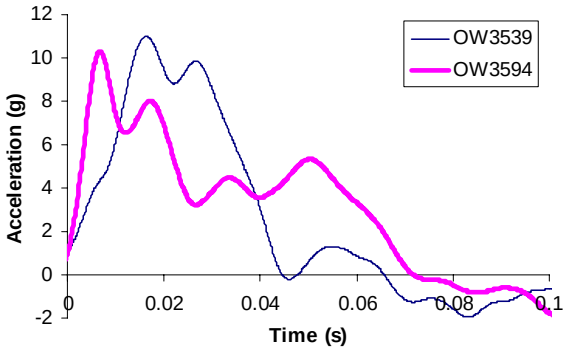

Figure 11.

The crash pulses from the OW3539 and OW3594, in tests at 100 % overlap with an impacting barrier generating a delta-V of 13 km/h to 13.3 km/h.

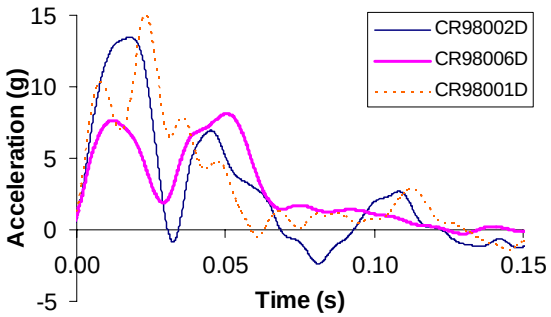

Figure 12.

The crash pulses from theCR98001, CR98002, and CR98006, in tests at 100 % overlap with an impacting rigid barrier generating a delta-V of 14.4 km/h to 16.9 km/h.

Real-World Rear Impacts

A large range of durations of crash pulses were found in the same type of vehicle where a similar delta-V was generated. Figure 13 and 14 and Table 3 shows the duration of the crash pulse, the mean acceleration and the delta-V from the real-world impacts from the vehicles T1 and T2. Furthermore, a considerable difference in shape of the crash pulse was registered in these cases (Figure 15 and 16). The duration of the pulses ranged from 77 ms - 134 ms. For vehicle T1, a change of velocity between 12.0 - 14.7 km/h and duration of the crash pulse from 77 ms to 109 ms was registered. For vehicle T2, a change of velocity between 17.1 - 20.4 km/h and duration of the crash pulse from 100 ms to 134 ms was registered.

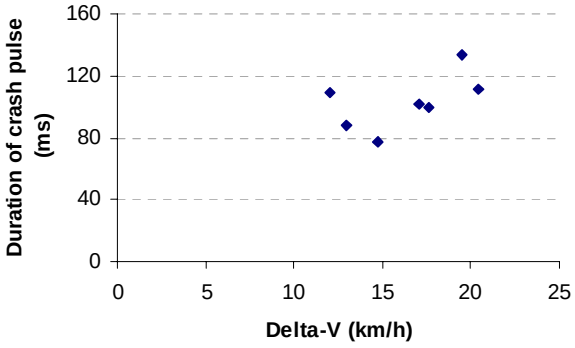

Figure 13.

The duration of the crash pulse and the delta-V from the crash recorder data from two different vehicle models of the same make.

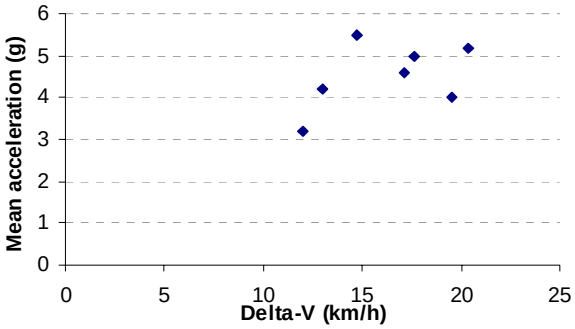

Figure 14.

The mean acceleration and the delta-V from the crash recorder data from two different year models of the same make and model of vehicle.

Table 3.
The duration of the crash pulse and the delta-V from the crash recorder data from two different year models of the same make and model of vehicle.

Car	CPR Number	Delta-V (km/h)	T_p (ms)	a_{mean} (g)
T1	C29521	14.7	77	5.5
T1	C30044	13.0	88	4.2
T1	C29614	12.0	109	3.2
T2	C30032	20.4	111	5.2
T2	C29732	19.5	134	4.0
T2	C29876	17.6	100	5.0
T2	C29739	17.1	102	4.6

Figures 15 and 16 shows the acceleration pulses from the real-world impacts from the vehicles T1 and T2.

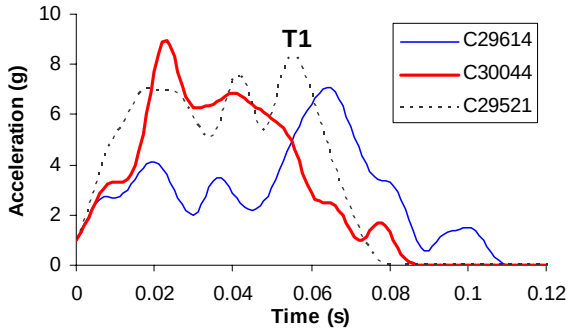

Figure 15.

The crash pulse measured in vehicle T1 in collisions with a change of velocity between 12.0 - 14.7 km/h.

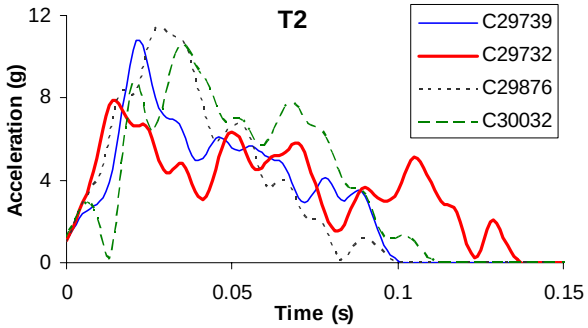

Figure 16.

The crash pulse measured in vehicle T2 in collisions with a change of velocity between 17.1 - 20.4 km/h.

DISCUSSION

A large variation in duration of crash pulse for a given delta-V and pulse shape can be produced in vehicles manufactured in the mid 1990s in rear impacts (Figure 6-12). Both delta-V and mean acceleration (i.e. duration of the crash pulse for a given delta-V) have been shown to influence the risk of AIS 1 neck injuries (Krafft et al., 2002). For a given delta-V a longer pulse will result in a lower mean acceleration and a lower risk of neck injuries (Krafft et al., 2002). The variation in durations of crash pulse for a given delta-V revealed in this study implies that vehicle seats aimed at reducing the risk of an AIS 1 neck injury should be designed in such a way that they provide the optimum protection in rear impacts in crashes where a great variation in duration of the crash pulse for a given delta-V might occur. These findings emphasise the importance of mean acceleration or the duration of crash pulse for a specific delta-V to be specified, in addition to delta-V, for sled tests that evaluate the protection from AIS 1 neck injuries of the seat, as suggested by Linder (2002).

A large variety of durations of crash pulse for a specific delta-V will be produced in the same car model, as exemplified by the real-world crash pulses collected from two year models of the same vehicle make and model (Figure 15 and 16). Therefore it can be expected that any vehicle will in real-world collisions be exposed for a large variety of durations of crash pulses for a specific delta-V. This might indicate that the design of the seat would have the largest potential to reduce the risk of AIS 1 neck injury in a rear impact since a huge variety of pulse

shapes will be generated in the same vehicle model due to the various configurations of the collisions.

In this study the duration of the crash pulse (T_p) was defined as the time when the acceleration changed from positive to negative after 90 % of delta-V had occurred. This definition was used to ensure that the main part of the energy was transferred into the impacted vehicle at T_p. From the crash pulses analysed for this study it was found to be a robust definition of the duration of the crash pulse.

The crash pulses were filtered with CFC 36 due to oscillations found in the crash pulses. It has been surmised that these oscillations may be due to the mounting methods used to attach the accelerometers to the vehicles. For the real-world data the oscillations could be due to the design of the crash recorder. The filtering of CFC 36 was chosen instead of the CFC 60 and did not influence the delta-V from any of the pulses (as exemplified in Figure 3). The benefit of the CFC 36 filtering was that it highlighted the main characteristics of the crash pulses and was thus the rational of the choice.

The two vehicles of the same make and model for the US and European market which were tested in this study had different bumper systems. The European bumper system (crush cans, bottom, Figure 17) was designed for the NCAR damageability test and required replacement after a test. The US bumper system (hydraulic shock absorbers, top Figure 17) resulted in no damages in both rear-into-flat barrier and rear-into-pole impact test at five mile per hour.

Figure 17.

The US bumper (upper) and the European bumper (lower) from the vehicle tested in test SL01001 and SL01002.

The US and European bumper systems resulted in similar shape of the crash pulse for the first 10 ms (Figure 9). After that the first peak acceleration was reach the shape of the two pulses developed somewhat differently in terms of when maximum and minimum magnitude of the pulses was reached.

The range of delta-V explored in this study cover the range where rear impacts causing AIS 1 neck injuries most frequently occur (Parkin et al., 1995, Hell et al., 1999, Temming and Zobel, 2000). The main part of the crash tests and real-world data were from delta-Vs at or close to those suggested as delta-Vs for sled tests that evaluate the protection from neck injuries in rear impacts. The delta-V for these sled tests has been proposed to 15 km/h or 16 km/h (Cappon et al., 2001, Muser et al., 2001 and Langwieder and Hell, 2002). For each vehicle in the crash tests a range of durations of the crash pulse for a specific delta-V according to various crash configurations as for the real-world data can be expected. The range of durations of crash pulses for delta-Vs at 14.9 km/h or 17.1 km/h would, according to the results shown in Figure 4 and 13, be at least 69 ms to 130 ms which correspond to a range of mean acceleration of 3.3 g to 7 g. The range of duration of the crash pulses published by Heitplatz et al. (2002) were for the delta-V of 15.7 km/h to 16.9 reported to be approximately 90 ms to 110 ms. These findings are within the range of what has been found in this study. And not surprisingly, with a larger number of vehicle tested the range of duration for a specific delta-V widens, as show in this study.

Mean acceleration has for frontal collisions been shown to influence the risk of injuries (Ydenius, 2002). In that study it was shown that increased mean acceleration increased the risk of MAIS 1 injuries. Of the MAIS 1 injuries in Ydenius (2002) neck injuries are approximately 30 % of these. As a consequence, Ydenius findings emphasises the findings in this study of the importance of mean acceleration with respect to neck injuries.

Recently, attention has been focused on the need to define an acceleration pulse for standardised rear impact testing to evaluate the risk of AIS 1 neck injuries. In sled test proposals (Cappon et al., 2001, Muser et al., 2001 and Langwieder and Hell, 2002) corridors for the crash pulses with a wide range of durations of the crash pulse for a specific delta-V has been suggested to be used. From the results of this study it is not possible to identify a typical mean acceleration (which correspond to a duration of the crash pulse) for a specific delta-V either in the laboratory crash tests or from the real-world data. It might be the case that in rear impacts with a risk of AIS 1 neck injuries there is not one typical pulse or

impact severity to be found. Rather a range of duration of crash pulses and delta-Vs that influence the risk of injury. Therefore it is suggested that duration of the crash pulse or mean acceleration, in addition to delta-V, should be specified for impact severity of sled test that evaluate the protection from the seat in rear impacts. This should be taken into consideration in such tests to minimizing the risk of sub optimization of seat protective performances.

CONCLUSIONS

From laboratorial tests with various vehicles impacted at the rear, a range of crash pulse durations between 65 ms to 130 ms was found for delta-Vs from 10.2 km/h to 19.4 km/h. Furthermore, from real-world rear collisions of the same vehicle make, a range of duration of crash pulse between 77 ms to 134 ms was found for delta-Vs from 12 km/h to 20.4 km/h.

This study shows that a similar delta-V can be generated by a variety of mean accelerations. Since mean acceleration have been found to be the main factor influencing the risk of AIS1 neck injuries, both delta-V and the duration of the crash pulse for a specific delta-V (i.e. mean acceleration) should be taken into consideration when defining impact severities in sled test procedure for vehicle seat safety performance assessment. In a sled test procedure a specification of a delta-V is therefore suggested to be accompanied with a specification of the mean acceleration or the duration of the crash pulse and the range of duration for a given delta-V of crash pulses that the seat could be exposed to, be taken into consideration in such tests.

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Evaluating the Uncertainty in Various Measurement Tasks Common to Accident Reconstruction

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ABSTRACT

When performing calculations pertaining to the analysis of motor vehicle accidents, investigators must often select appropriate values for a number of parameters. The uncertainty of the final answers is a function of the uncertainty of each parameter involved in the calculation.

This paper presents the results of recent tests conducted to obtain sample distributions of some common parameters, including measurements made with tapes, measurements made with roller-wheels, skidmark measurements, yawmark measurements, estimation of crush damage from photographs, and drag factors, that can be used to evaluate the uncertainty in an accident reconstruction analysis. The paper also reviews the distributions of some pertinent data reported by other researchers.

INTRODUCTION

When performing calculations pertaining to the analysis of motor vehicle accidents, investigators often must establish values for a number of parameters, such as drag factor, distances along roadways, crush depths, skid lengths, and yaw marks. Some of these are established by making measurements, some through the use of tables, and others by using judgement. The uncertainty of the final answers is a function of the variations of each parameter involved in the calculation. Though it has long been recognized that variations exist in all measured data, including those related to accident analysis [1, 2, 3, 4] very little published information exists to assist the investigator in assigning realistic input parameter variations.

There are at least three different ways to estimate the uncertainty of reconstruction calculations. One approach is simply to combine the parameter ranges in such a way as to generate extreme high and low results. Though possible, this situation is unlikely to exist in the real world. A more analytical approach requires taking partial derivatives of the constitutive equations involved in the analysis. With these derivatives, the overall uncertainty of a calculation can be estimated using the Root-Sum-of-Squares (RSS) method outlined by the NIST [5] and demonstrated by Tubergen. [6] However,

given the complicated nonlinear equations typically used in accident reconstructions, this can become impractical. Another approach, Monte Carlo simulation, often provides a more convenient means of quantifying the combined uncertainty. Monte Carlo analyses require the assignment of probability distributions and their parameter values to the input variables in order to determine the uncertainty of a reconstruction result to a particular confidence level. Several researchers have demonstrated the application of Monte Carlo Simulation techniques in accident analysis [2, 6, 7,8], but none has discussed which probability distributions should be used, nor have they suggested ranges that should be used for variations that exist in commonly used parameters.

This paper presents the results of recent tests conducted to obtain sample distributions of some common parameters, including measurements of well-defined distances made with tapes, roller-wheels, and a laser transit, measurements of skidmarks, arcs, and yawmarks, measurement of crush damage, estimation of crush damage from photographs, and measurement of drag factors by several methods. This paper also reviews the distributions of pertinent data reported by other researchers in the accident reconstruction field. Prudent application of proper distributions and ranges can lead to more accurate accident reconstructions, regardless of which approach is used.

STATISTICS AND PROBABILITY DISTRIBUTIONS

Notation in this paper follows proper statistical practice in that $\bar{X}$ and s are used to indicate the mean and standard deviation of a sample (comprised of a set of measurements) and μ and σ are the mean and standard deviation of a population.

After systematic errors and blunders have been accounted for in a group of measurements with one variable, random measurement errors will remain. In practice, random errors often follow a normal distribution, or "bell-curve." The frequency function describing this type of distribution is written as:

$$f(x) = \frac{e^{-(x-\mu)^2/2\sigma^2}}{\sigma\sqrt{2\pi}} \qquad \text{EQ. 1}$$

Where μ = population mean
σ = population standard deviation

In a normally distributed population, the measured value falls within one standard deviation of the mean value ($\mu \pm \sigma$) 68.3% of the time, as shown in Figure 1. The measured value can be expected to fall within two standard deviations of the mean ($\mu \pm 2\sigma$) 95.5% of the time and within three standard deviations ($\mu \pm 3\sigma$) 99.7% of the time. [9] There is no absolute upper or lower bound to the value. This function has a total area under the curve from $-\infty$ to $+\infty$ of 1 square unit. Unless

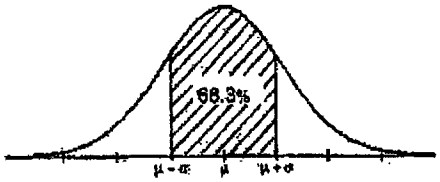


Figure 1: Normal distribution, showing the area encompassed by the mean plus or minus one standard deviation ($\mu \pm \sigma$)

otherwise stated, all statistical analyses in this paper will assume a normal distribution.

Two ways to determine if a particular set of data follows a normal distribution are the use of a normal probability axis and a Chi-squared "goodness-of-fit" test. In the former method, one determines if the data follows a straight line when plotted on a normal probability axis, as shown in Figure 7. The latter test is used to determine to a particular level of confidence if the data is a sample from a normal distribution. A comprehensive description of this method is beyond the scope of this paper, but can be found in most statistics books such as reference [10].

A second type of distribution commonly encountered in accident reconstruction analyses is the uniform or rectangular distribution. This type can be used in the absence of evidence to suggest that the value is more likely to be near the center of the range. This distribution is more conservative than a normal distribution, as it gives equal probability to all values in the specified range. At the same time, it precludes values outside the specified range, so the bounding terms, (μ - a) and (μ + a), must be selected with great care. The standard deviation for this type of distribution is equal to [*a / SQRT(3)*]. The range covered by ($\mu \pm \sigma$) includes 57.74% of all values. [10]

For a review of the terminology of uncertainty and statistical methods associated with evaluating uncertainty in measurements and calculations, the reader is directed to the references. [1, 5, 10]

LINEAR MEASUREMENTS TASKS

A series of linear measuring tasks were devised to test the repeatability of measurements that required little or no judgment regarding the location or dimensions of the item being measured. Uncertainty from these measurement tasks forms the basis for better understanding the errors involved in more complex tasks.

Many of these measurement tasks were conducted during the World Reconstruction Exposition 2000 (WREX2000) held in September 2000, at College Station, Texas, USA.

fairly consistent (within an 8-inch span) while the selection of the mark's beginning point was less consistent. Table 5 contains the descriptive statistics for the measurement results. The measured length of the lighter rear tire mark was observed to decrease as the day progressed, while the measured length for the fairly dark front mark did not change significantly. Results are summarized graphically in Figure 16.

	Front+Rear ft. (m)	Front mark ft. (m)
Minimum	72.0 (21.9)	64.0 (19.5)
Maximum	77.4 (23.6)	68.6 (20.9)
Mean	75.2 (22.9)	67.0 (20.4)
Standard Deviation	1.7 (0.5)	1.7 (0.5)
Count	14	10

Table 5: Descriptive statistics of skidmark measurements.

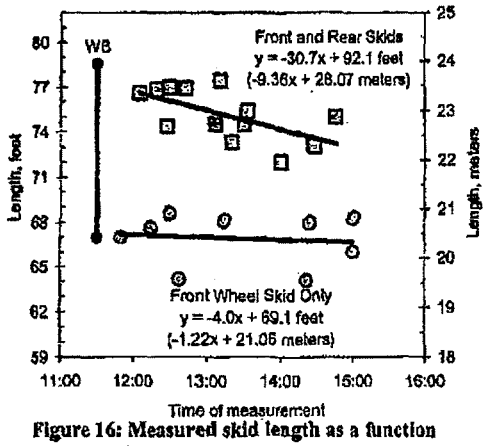


Figure 16: Measured skid length as a function of time of day. Trendlines use "x" equal to the fraction of the day, noon=0.5.

MEASURING VEHICLE CRUSH

The amount of deformation a vehicle sustains is often of interest to a reconstructionist conducting an energy-based accident analysis. While the vehicle itself is sometimes available for measurement, reconstructionists are occasionally asked to determine deformation depths from one or more photographs. Experiments examining the variation involved in each of these methods of crush measurement and estimation are described below.

DIRECT MEASUREMENT OF DAMAGED VEHICLE

Participants were provided a Chevrolet Astro van, shown in Figure 17, which had been involved in a partial overlap frontal impact. They were given a data sheet (Shown in APPENDIX A) on which to record their measurements which included a description of the

Figure 17: Damaged Astro-van with measurement jig for direct crush measurement experiment.

CRASH 3 measurement protocol [21], a description of the parameters to be measured, the vehicle's original overall length, overall width, and wheelbase.

The equipment made available was an adjustable, rectangular frame on the ground that could be used as a reference, if desired. Two measuring devices were provided: a 16-foot (5-m) steel rollup tape measure and a 6-foot (2-m) folding carpenter's ruler. A plumb bob was also available.

A review of the data showed that three participants recorded the data backwards. After reversing the order of their data points, the 17 crush profiles are shown in Figure 18. Using a weighted average which considers only half the value of the end points, the average crush depth recorded by the 17 participants ranged from 11.8 to 34 inches, with an average of 19.4 inches and a standard deviation of 5.2 inches. The reported length of the damage area ranged from 45 to 78 inches, with an average of 62.4 inches and a standard deviation of 9.9 inches.

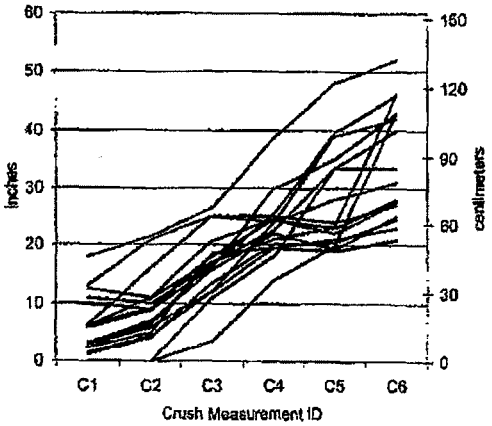


Figure 18: Residual crush measurement profiles recorded by 17 participants.

CRUSH ESTIMATES FROM PHOTOGRAPHS

Participants were asked to estimate the crush and or Equivalent Barrier Speed (EBS) of a vehicle from a single photograph (Figure 19) or a set of two photographs (Figures 20a, and 20b) without any additional information. In the single-photograph exercise, 57 participants provided 11 crush estimates and 49 EBS estimates. The average estimated crush was 13.6 inches with a standard deviation of 4.2 inches. The EBS results are given in Table 6.

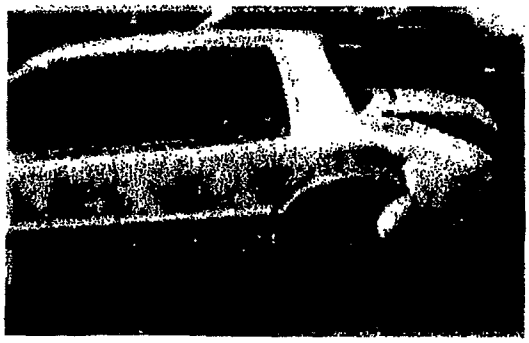


Figure 19: Single photograph used for speed/crush estimate.

EBS, mph (kph)	# of responses
6 – 10 (9.7-16.1)	2
11 – 15 (17.7-24.2)	12
16 - 20 (25.8-32.2)	16
21 - 25 (33.8-40.3)	14
26 - 30 (41.9-48.3)	5

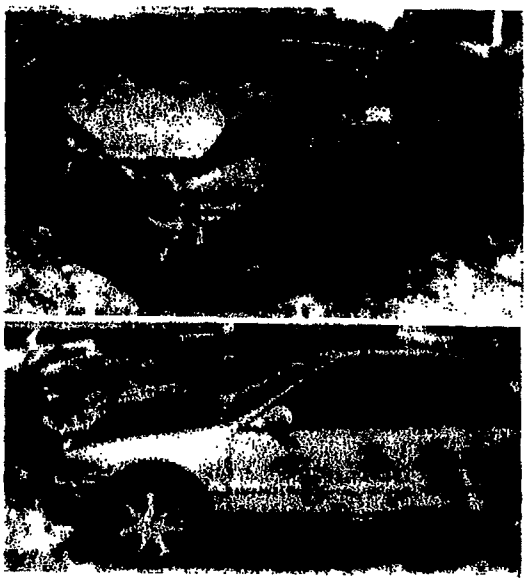
Table 6: Crush estimates from a single photograph.

In the two-photograph exercise, 52 participants provided 8 crush estimates and 51 EBS estimates. The average estimated crush was 13.1 inches with a standard deviation of 3.3 inches. The EBS results are given in Table 7.

EBS, mph (kph)	# of responses
11 – 15 (17.7 - 24.2)	5
16 - 20 (25.8 - 32.2)	12
21 - 25 (33.8 - 40.3)	18
26 - 30 (41.9 - 48.3)	12
31 – 35 (49.9 - 56.4)	3
36 – 40 (58.0 - 64.4)	0
41 - 45 (66.0 - 72.5)	1

Table 7: Crush depth estimates from two photographs.

Most of the participants in this exercise reported that they would not attempt to use information generated in this fashion in a reconstruction without additional details and analysis.



Figures 20a and 20b: Two photographs used together for one crush estimation exercise.

CONCLUSIONS

This paper presents the results of a variety of experiments designed to quantify the uncertainty in some measuring tasks commonly faced by accident reconstructionists. Modelling the probability of many accident reconstruction variables as normally distributed appears to be appropriate. The standard deviations from many common measurement tasks were presented. These data can help determine appropriate ranges for variables used in accident reconstruction uncertainty analyses.

When making measurements that have been shown here to have very small standard deviations, an investigator may be comfortable making a single measurement. When making measurements that have been shown to have large standard deviations, though, an investigator may wish to consider making several repeated measurements to generate a distribution from which a centered value can be selected.

In the one measurement task with a large standard deviation in which the root causes were studied (drag sleds), lack of standardized training was found to be a significant issue that affected the tool's repeatability. It seems apparent from the results presented in this paper that repeatability of some other tasks common to accident reconstruction would improve with standardized protocols.

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Analysis of Human Test Subject Kinematic Responses to Low Velocity Rear End Impacts

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ndible/maxilla. There was also a photographically visible ark placed below and slightly behind the left external auditory canal over the mastoid prominence as an approximation of the lateral projection of the upper end of the cervical spine. Reference marks were applied to the skin over the test subject's left neck, simulating the lateral projection of the cervical spine. Targets were placed approximately over the left gleno-humeral joint and lateral left elbow on a tight fitting garment worn over the torso and arms. A Hybrid III anthropomorphic test dummy was fitted with a biteblock type accelerometer assembly and had similar right side anatomical reference point markings applied, with the exception of the dummy's already exposed neck area.

INSTRUMENTATION — Each vehicle, had a triaxial LSCB-10 accelerometer array mounted on the vehicle frame to measure Gx (forward/rearward), Gy (right and left lateral), and Gz (upward/downward) motions and a biaxial accelerometer array (Gx and Gz) on the driver's side seatback. Contact switch operated flash units were installed in visible positions to allow photographic time marking of initial bumper to bumper contact and a similar contact switch cued the electronic data acquisition system. Additional instrumentation to accomplish other test objectives was also installed on the vehicles. Test subject instrumentation included a lightweight triaxial accelerometer assembly of Endevco #7290-30 and ndevco #7290-10 accelerometers mounted on a short uminum strip fixed to an individually fabricated mouth piece (biteblock) which, when held with normal jaw closure pressure, allowed no appreciable relative motion between the accelerometer assembly and the test subject's maxilla/mandible. An identical accelerometer array mounted on an aluminum strip was affixed in an equivalent position on the Hybrid III manikin's head. A biaxial accelerometer assembly using similar sensors was affixed to a corset-like garment and was worn by one test subject during two of the test runs to measure Gx and Gz direction acceleration. Electronic data transfer during test runs was accomplished by a sliding loop umbilical bundle connected to a PAC-5800 high volume data acquisition system housed nearby.

PHOTOGRAPHIC EQUIPMENT — Photographic documentation of test runs was accomplished by several Redlake LoCam Model #51 high speed 16 mm cameras operated at 500 frames per second (nominal) and equipped with an LED timing light operating at 100 hertz. These cameras were mounted at various locations on the vehicles and from several fixed positions about the test site. One high speed video unit and several tripod mounted standard video cameras also recorded the events.

TEST SITE — The test site was established on a level section of a standard, asphalt paved roadway and had electronic speed trap instrumentation and high speed video king with near realtime velocity measurement capability. mpact speed reproducibility was achieved by the use of a specially constructed ramp permitting gravity acceleration of the striking vehicle. The striking vehicle's starting position on the ramp was calibrated before each test run to ensure that the resulting velocity at the impact point was in the desired range. The actual closure speeds and resulting changes in velocity of both the striking and the struck vehicles during the test runs were accurately determined by high speed film, high speed video and the electronic speed trap with satisfactory agreement. High speed cameras on the vehicles and at fixed site positions were actuated by a central electronic control and the video cameras were controlled by individual operators responding to auditory and visual cues.

TEST PROCEDURES — Test runs were conducted according to a protocol which tested a variety of combinations of vehicle to vehicle collisions and exposed test subjects to both striking and struck roles during tests planned for a forward struck vehicle ΔV of 4 kph (2.5 mph) and 8 kph (5.0 mph). The striking vehicle was backed up the ramp by the test subject-driver to a calculated position and released with the transmission in neutral and the engine running. It then rolled down the ramp and through the impact point speed trap where the velocity was recorded. This procedure was repeated until the desired impact speed was reproducibly achieved. The vehicle to be struck was then placed into its stationary position at the impact point. The striking vehicle then rolled down the ramp and over the level pavement to the impact point. No vehicle control inputs were made during test runs, except for minimal steering inputs to ensure centerline contact between vehicles and late braking after the test impact perturbations were over. In some cases, to prevent an over or underride situation, the height of one of the two vehicles was elevated by the use of wooden planking which formed an elevated roadbed. After each test collision, the driver's physical condition was checked, post-test photographic assessment of vehicle damage was completed, and electronic test result data storage was accomplished. Data from ten manned vehicle to vehicle test collisions were recorded.

HEAD, NECK AND TRUNK KINEMATIC RESPONSE ANALYSIS — The purpose of this article is to report our findings after a detailed analysis of human head, neck and trunk kinematic responses occurring during and immediately after low velocity rearend impacts. Relevant data included the recorded G-time information from the human and vehicle mounted accelerometers, displacement-time data taken from the high speed film record and the slow motion video record of each test subject's motion during the collision sequences. The electronic G measurement data was processed into a usable form with smoothing to eliminate noise artifacts and very short term transients. Biteblock G-time vector resultants were calculated from Gx, Gy and Gz data recorded for each collision sequence. In order to obtain true (earth orthogonal) G vector resultant data, mathematical coordinate transformation was done to account for the curvilinearly displaced path of the biteblock reference frame from its initial earth orthogonal orientation and required utilization of time-angle data obtained by measurement from the high speed film record. An additional series of mathematical manipulations gave an earth reference based G-time history for a point near the junction of the head and upper cervical spine. This information was correlated with point displacement information obtained from the high speed film by plotting each

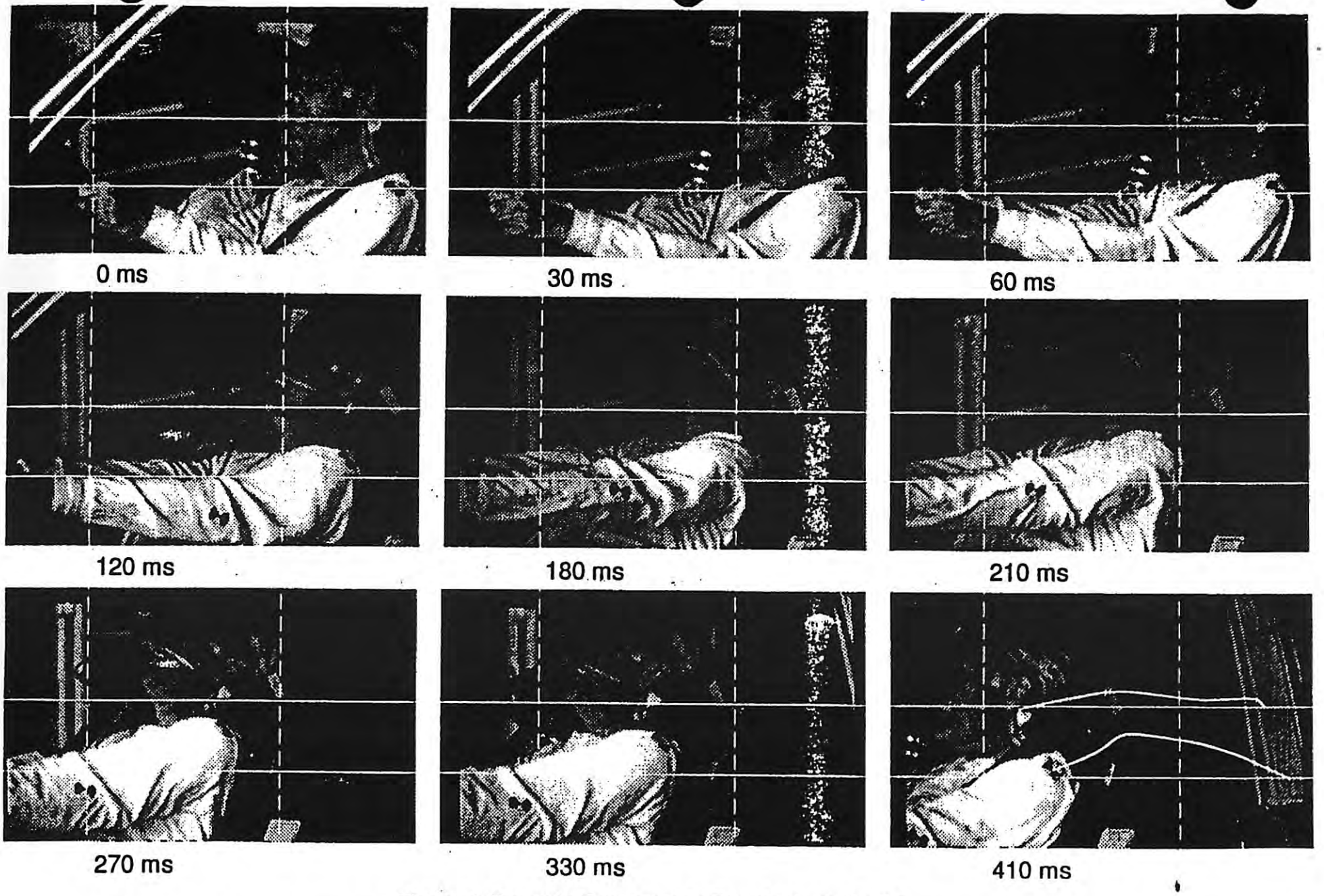

Figure 1. Example Response to Rear Impact - Test 7
Note. Grid lines are earth fixed. Last picture shows time trace of neck top and shoulder pathways.

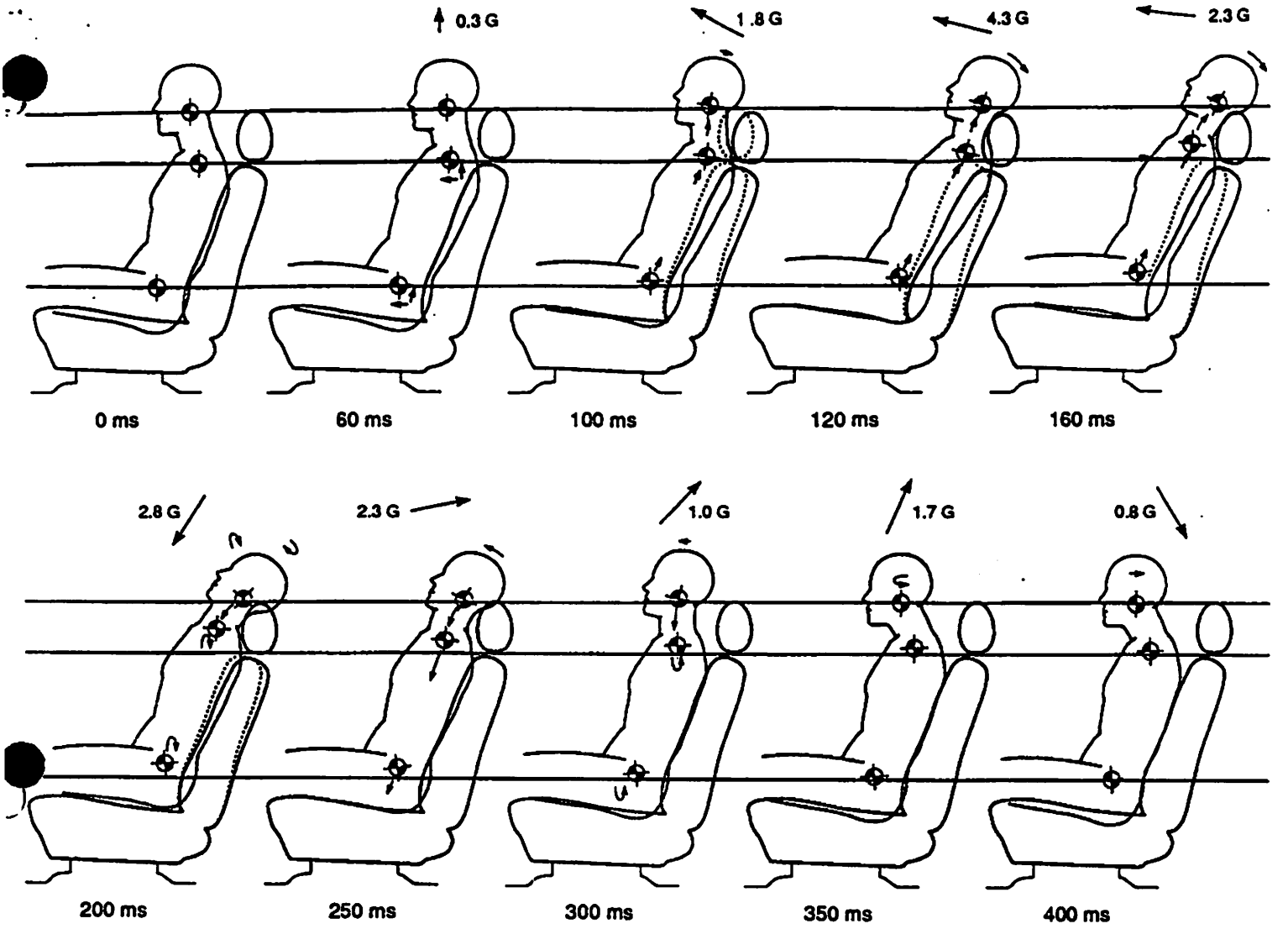

Figure 2. Head, Neck and Trunk Responses to Low Velocity Rear End Impact

subject's head and neck were still almost stationary with respect to the earth until about 120 milliseconds when, as the subject's hips and trunk rose upward on a path parallel to the rearward flexed seatback, his neck appeared to be axially compressed and straightened as the top of the cervical spine began moving upward and rearward with respect to the forward moving vehicle. Subsequently, the subject's head began a biteblock upward and rearward rotating movement with respect to the subject's shoulders. By 160 milliseconds the forward and upward movement of the subject's ascending upper torso had begun to pull the base of his neck forward into apparent tension and starting the forward motion of the subject's head, even as his occiput continued to tip downward towards the seat headrest.

Phase 3 - Head Overspeed/Torso Recovery (200 to 300 milliseconds) — At 200 milliseconds the upward motion of the subject's trunk and shoulders had ceased after about 9 centimeters (3.5 inches) of rise and the extension and rotational angulation of his head had stopped about 45 degrees rearward from vertical. The subject's top of the cervical spine marker point had risen about 1.25 centimeters (0.5 inches) above its initial vertical position and the subject's head was starting to reverse its motion, with respect to the vehicle, into a forward arcing movement. By 250 milliseconds the forward rebounding head had not yet reached vertical, but the subject's trunk, neck and head were already descending along a path parallel to the seatback. His trunk was nearly halfway towards its starting position with respect to the seat bottom. By this time, the seatback had returned to its pre-impact normal angle and the subject's torso was rebounding forward and away from the seatback's surface. At this point the test subject's upper body was probably being actively retarded by the tightened restraint system. The restraint system had become more than normally tightened when the spring powered seatbelt retractor reeled in about 5 to 8 centimeters (2 to 3 inches) of seatbelt and shoulder harness slack that had

been produced by the initial compression and relatively rearward flexion of the forward moving seatback cushion by the stationary test subject's torso. This autotightening phenomenon was noted consistently in several vehicles during our test series.

Phase 4 - Head Deceleration/Torso Rest (300 to 400 milliseconds) — After 300 milliseconds from first bumper contact, the descent of the test subject's trunk had been completed and his trunk was moving at essentially the same velocity as the vehicle. The rebound forward motion of his head, now positioned near vertical, continued but was being actively decelerated by the tension in his neck. At about 400 milliseconds his head had reached its most forward position with respect to the vehicle. The biteblock to mastoid reference line was nearly level with the horizon and the head was slowing to a nearly level, forward and lowered position with respect to the subject's shoulders. The subject's head then began a return movement relatively rearward and upward towards a normal upright position over his shoulders.

Phase 5 - Restitution Phase (400 to 600 milliseconds) — After about 450 milliseconds after first bumper contact, all test subject body parts were traveling at approximately the vehicle's velocity and his immediate impact related motions were nearly completed. During this period the test subject's head and biteblock returned to approximately their pre-test positions with respect to the vehicle. However, the test subject's shoulders and hips rested in a position about 3.8 centimeters (1.5 inches) higher than before.

TIME-DISPLACEMENT AND ACCELERATION ANALYSIS — Analysis of the time-displacement record of the body reference targets and the G-time record of the biteblock sensors, referenced by calculation to a point approximately at the top of the cervical spine, defined the x direction acceleration profile for each test. Analysis also confirmed the existence of significant upward and downward accelerated motion of the trunk, neck and head along the z axis. Vehicle and test subject motions along the y axis were not found to be significant in this test series.

Test subject cervical extension and flexion angles observed during this test series were always found to fall within the subject's voluntary physiological limits. Hyperextension or hyperflexion did not occur during any of the test runs. The maximum cervical extension (nose up) observed for all struck vehicle test runs appeared to be self limited to a maximum of about 40 to 45 degrees, even for test runs using the van, which had no headrests. Rebound cervical flexion was minimal for every test with head angulation (nose down) from the normal upright posture averaging three to five degrees or less, as the typical test subject's head followed a mild, head level, controlled forward and downward decelerative motion, followed by a gentle return to a normal upright posture. Throughout their impact related kinematic responses, each test subject's head and torso generally remained within 13 to 18 centimeters (5 to 7 inches), or less, of their pre-impact positions with respect to the vehicle.

Figure 3 shows Test Run 7 resultant accelerations acting at the top of the cervical spine for selected times, beginning 60 milliseconds after bumper contact (time 0). Figure 2 also shows these G vector resultants.

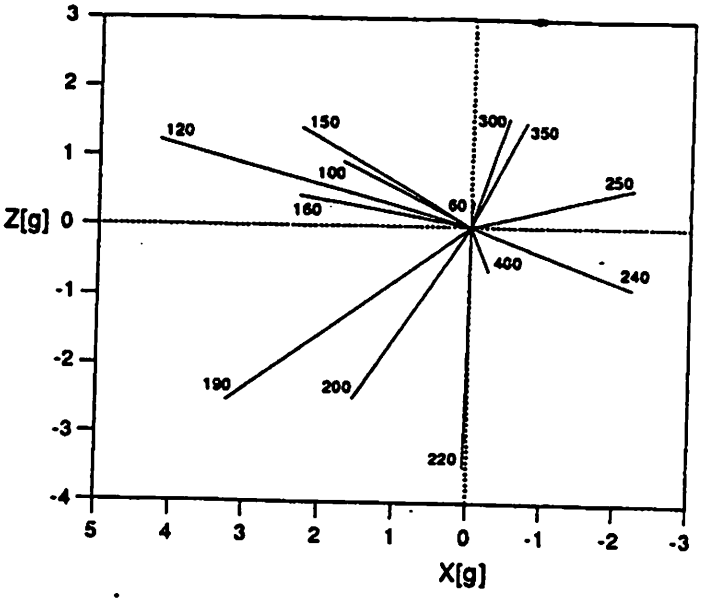

Figure 3. Resultant Acceleration (Gs) of the Neck Top at Selected Times after Impact (milliseconds). Test 7

Phase 1 - Initial Response (0 to 100 milliseconds) — The initial top of the cervical spine resultant acceleration at about 60 milliseconds after contact is upward at about 0.3 G and soon rises to 1.8 G upward and forward by 100 milliseconds.

Phase 2 - Principal Forward Acceleration (100 to 200 milliseconds) — At about 120 milliseconds acceleration forward and upward climbs to an overall peak of about 4.3 Gs. As forward Gx acceleration continues, the top of the cervical spine Gz acceleration reverses from a +1.2 Gz positive (upward) peak at 150 milliseconds to a peak negative (downward) -3.5 Gs at about 220 milliseconds. At 190 milliseconds resultant acceleration at the top of the cervical spine is nearly 4 Gs in a downward and forward direction.

Phase 3 - Head Overspeed/Torso Recovery (200 to 300 milliseconds) — At 200 milliseconds, the subject's head begins to arc forward with respect to his torso and over the next 30 milliseconds the top of the cervical spine is being accelerated forward and downward at 2 to 3.5 G. Simultaneously, the torso begins to return forward and down the seatback slope. By 220 milliseconds most of the forward acceleration of the top of the cervical spine has been accomplished and the actual upward motion of the torso and head has nearly ceased. Also by this time, the acceleration altered seatback angle has nearly returned to its normal position and the seat springs have become almost unloaded. By 250 milliseconds, the forward moving torso is being retarded by the restraint system and the Gz acceleration of the

top of the cervical spine is reversing as the trunk is now about halfway down the seatback and moving towards its original position. The subject's head has now arced forward with respect to the torso, decreasing its rearward angle to about half the maximum angle achieved and the top of the cervical spine is being accelerated rearward and slightly upward at about 2.3 G. Over the next 50 milliseconds, as the seat cushion is compressed by the subject's downward moving body, the upward Gz acceleration experienced at the top of the cervical spine increases to about 1.5 G.

Phase 4 - Head Deceleration/Torso Rest (300 to 400 milliseconds) — Around 300 milliseconds the downward motion of the trunk reverses and the subject's head has returned to about its pre-impact position over his torso. At this point, the resultant acceleration at the top of the cervical spine decreases to about 1 G directed upward and rearward. During the next 50 to 100 milliseconds, the trunk and torso begin to reassume a rest position. At the same time, the more rapidly forward moving head continues forward and downward while maintaining a nearly level position with respect to the horizon. During this maneuver the lower neck becomes mildly flexed while the top of the cervical spine mildly extends resulting in a kind of "level head bob". At around 400 milliseconds the subject's head begins a controlled return upward and rearward while still remaining nearly level. The resultant upward and rearward acceleration at the top of the cervical spine at this point is 1.7 G.

Phase 5 - Restitution Phase (400 to 600 milliseconds) — After 400 milliseconds the test subject's body position returns to about its pre-impact position with the exception of hip and shoulder positions about 3.8 centimeters (1.5 inches) higher than at the start of the test. This may have resulted from shifting of trunk and hips on the seat's surface, residual spinal curvature straightening, or perhaps due to lack of time for the seat cushions to fully conform to their recently reacquired load. Between about 400 and 600 milliseconds after initial contact, mild Gx and Gz oscillations appear to briefly continue but are rapidly damped and soon blend into a low level "jiggle", probably associated with the vehicle's movement over the pavement.

GENERALIZED FINDINGS

The kinematic responses of the struck vehicle test subjects for each test run were qualitatively similar to the above description. There were some subtle but consistently observed differences in the kinematic responses of test subjects due to the dissimilar design between the coupe/convertible seats and the more upright and somewhat stiffer seats of the pickup truck and van. The test subject's kinematic responses for the 4 kph (2.5 mph) ΔV test runs were similar to the 8 kph (5.0 mph) ΔV categories but visibly reflected the four-fold decrease in collision related energy. Lower velocity test runs, in general, were associated with considerably lessened overall kinematic activity and much milder cervical extensions. From a clinical standpoint, the nominal 8 kph (5 mph) ΔV test runs appeared to be on the threshold for mild cervical strain injury for our repetitively exposed test subjects. The nominal 4 kph (2.5 mph) ΔV test runs were considered later by the participating physician test subjects to have been so very mild that a single exposure would have been unlikely to have resulted in any symptomatology.

DISCUSSION

In reviewing the voluminous literature on this subject, especially the many articles on clinical analysis, experimental testing and computer based modeling efforts, one must be careful about making the assumption that the conclusions about human head and neck kinematics reached in these studies necessarily apply to low and very low velocity rearend collisions involving "real people". The majority of these studies have been primarily based on higher speed, 24 to 80 kph (15 to 50 mph) or more, rearend collisions utilizing dummies, cadavers, animals, computer models and very few live volunteers.

The puzzling, and sometimes stubbornly persistent clinical symptom picture frequently associated with "whiplash" injury, especially in the absence of objective physical findings, leads by default to the assumption that something unknown is happening during low velocity rearend collisions which is damaging cervical structure(s) without causing objectively discernable acute changes. Since exaggerated neck motion far beyond tolerable human limits had been frequently observed in dummies and cadavers during high speed testing, it has been commonly assumed that cervical hyperextension and hyperflexion would also occur during low and very low velocity collisions. It has been conjectured by many that the forced movement of the neck beyond physiologic limits was the injury mechanism causing the "whiplash" syndrome, especially in thin necked, unprepared people.

The test subjects in this series were robustly healthy middle-aged men who were well aware of testing procedures. To the maximum extent possible, test subjects were kept unaware of the specific time of the impending test impact and all consciously attempted to maintain a normal relaxed muscle tone while awaiting the impact. While no test subject exceeded his normal voluntary range of cervical motion during the test runs, three of the four did subsequently develop transient, mild cervical strain symptoms which indicated that there was an injury mechanism that was not dependent upon exceeding the physiologic limits of cervical motion.

There has been an occasional reference in the reviewed literature to vertical movement of the rearended vehicle test subject (usually a dummy) under specific test circumstances, or as a possibility in conceptual modeling. However, since forward acceleration becomes so dominant in the often studied higher speed rearend collision, analysis of test subject vertical motion has been generally ignored. We were conceptually aware before the test series of the possibility of impact-related acceleration in the Gz direction and made provisions to measure it. However, the magnitude and apparent causes of the vertical movement associated with our human test subjects and the corresponding lack of a

similar vertical excursion observed in the Hybrid III dummy had not been predicted from our review of the subject literature.

Observation of the high speed film record of the struck vehicle test runs disclosed that the test subject's early upward neck movement and initial forward head motion were due to several probable mechanisms. First, there was an upward lofting action caused by the normal vehicle seatback angle, which was itself increased by acceleration related deflection of the seatback. Second, there appeared to be an acceleration related straightening of the normal thoracic, cervical and, perhaps, lumbar spinal curvatures against the forward moving seatback surface, resulting in an apparent axial lengthening of the spine. Third, resistance to neck structure bending generated by the normal postural muscle tone of the upright neck may contribute to the initial forward head acceleration as recorded at the top of the cervical spine.

The upward movement of the test subject's trunk and neck base caused a more complex than expected motion of the neck and head. This initial upward motion was immediately followed by a sudden and surprisingly vigorous descent of the trunk, which may have resulted from a "rubberband" effect of the kinematically straightened and stretched trunk/spinal structures as the upwardly moving pelvis becomes restricted by the lapbelt. Shortly after the subject's trunk reaches the limit of upward compliance permitted by the lapbelt, the spine appeared to forcefully return to its original unstretched and curved state.

According to our measurements of the struck vehicle test subjects during the 6 to 8 kph (4 to 5 mph) ΔV test runs, there was an initial axial compression, measured at the top of the cervical spine, which accelerated the head at about 1 to 1.5 Gs upward, followed, as the base of the neck was pulled forward and downward, by a brief peak axial tension through the rearward extended cervical spine of about 2 to 4.5 Gs. The less energetic 3 to 4 kph (1.9 to 2.4 mph) ΔV test runs resulted in essentially the same type of head and neck motion with much decreased accelerations and kinematic responses.

At the low and very low velocity rearend collisions evaluated during our test series, the most likely injury to have occurred was a mild cervical and upper shoulder muscular strain. In our experience, this relatively mild injury would have been expected to be, and was, self-limited. It appears from our present results that the principal mechanical stress on the cervical spine related to rearend vehicle collisions in the low to very low velocity range is a rapid compression-tension cycle directed axially through the cervical spine and neck musculature as the neck sequentially compresses, extends, accelerates the head in tension and then flexes, all occurring within the normal physiologic range of motion limits of the neck. A suggested probable injury causation mechanism consistent with these observations would be a localized cervical/upper thoracic muscular strain caused by rapid, short [illegible]rsion, forced muscular compliance to sudden unanticipated [illegible]ion, occurring at an onset rate beyond the physiologic olerance of the affected muscles' intracellular micro-tructures. Belated, overactive or out of synchronization reflex muscular tensioning to control head motion may contribute to the muscular strain process, although our test subjects muscular corrective mechanisms appeared to be appropriately coordinated and effective by about 200 to 250 milliseconds after impact. An additional, less likely, but possible injury mechanism may be simple compression-related acute micro-contusional injuries to the cervical and upper thoracic spine connective tissue and joint structures. Similar compression-tension injury causation mechanisms may account for the mild low back discomfort symptoms that are sometimes reported by individuals involved in low velocity rearend collisions.

Photographic observation of the head, neck and upper torso relationships that occurred during each test subject's peak cervical extension suggested the possibility of an inherent, anatomically based, neck extension limitation mechanism. A triangular shaped support structure for the top of the cervical spine is formed by the supporting structures of the upper torso, the partially extended (straightened) cervical spine and the muscles of the anterior neck that normally maintain the head erect. Initial cervical muscle bracing would involve pre-existing normal neck muscle tone, followed during the middle and end of the initial kinematic response period by reflex and voluntary muscular head righting activity. This progressively more active bracing mechanism, in conjunction with the downward and forward movement of the shoulders and base of the neck, (which occurred in our test series well before the subject's head and neck had reached full extension), may help self-limit (at least in the low velocity situation) neck extension to within physiologically tolerable limits. The absence of this active bracing mechanism in both anthropomorphic dummies and cadaver test subjects may account for the observation of unchecked rearward head motion and neck hyperextension reported in experiments utilizing these imperfect human analogs as test subjects.

CONCLUSIONS

The data from our low velocity rearend collision test series using volunteer test subjects supports the preliminary conclusion that substantial Gz direction acceleration occurs and is associated with both compressive and tensile forces sequentially directed axially through the cervical spine.

These push-pull forces probably represent an injury causation mechanism independent of the commonly described cervical "whiplash" hyperextension/hyperflexion mechanism. For rearend collisions within the velocity range included in our test series, the classic "whiplash" injury mechanism, seems unlikely since no hyperextension or hyperflexion was observed in any of our test subjects. Despite having experienced no neck excursions beyond their voluntary range limits, three of our four test subjects transiently had very mild, but clinically classic neck discomfort symptoms.

During the lower energy level 4 kph (2.5 mph) ΔV test runs, the test subject's relatively rearward head motion was similar but much milder and, in each case, the back of the test subject's head did not reach the headrest. The injury

causation potential during these tests was subjectively judged by the physician test subjects to have been minimal or non-existent.

The reported results of this low velocity test series suggest a compression-tension injury causation mechanism which probably can cause self-limited minor cervical, thoracic and lumbar muscle strains and, possibly, connective tissue and/or vertebral joint micro-contusional injuries and that may account for the discomfort symptoms commonly reported after low velocity rearend collisions. The very mild discomfort symptoms experienced by our three test subjects, after multiple test exposures, indicated that the 6 to 8 kph (4 to 5 mph) struck vehicle ΔV test conditions were probably at, or near, typical human threshold for very mild, single event musculoskeletal cervical strain injury.

The test results from our small number of test runs and relatively homogeneous test panel should be supplemented by further testing. This testing, which should include a wider variety of test subjects arranged in different seating positions, various riding postures and restraint system usage, would better define the complete range of expected kinematic responses by the vehicle riding general public to low and very low velocity rearend collisions.

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Advances in the Understanding of Rear Impact Collision –

Updating Physics, Biomechanics and Statistics

By John Smith, P.E. and Christina E. Smith

The intent of this article is to update the reader on advances in the understanding of rear impacts. While none of the information in the previous article, "The Physics Biomechanics and Statistics of Automobile Rear Impact Collisions[1]" is incorrect, the last 16 years have seen significant advances in the understanding of how and why people are hurt in rear impacts. This article does not necessarily repeat data from the original but expands upon it as appropriate.

Over the past 16 years, significant research has occurred in the area of rear impact collisions. The direction of the research has tended to follow one of two paths, although overlap does exist. Researchers based the first course of study on applying the principles of engineering and biomechanics to accepted research techniques to understand better what was occurring in rear impacts. The second approach was litigation driven and was often predicated upon pseudo science or actual science distorted[2] to advocate a position. In this paper, the current state of the applicable science regarding an area is addressed followed by a discussion of many of the common errors promulgated in litigation.

Physical Response of the Automobile

Over the last decades significant changes have occurred in automobiles. Sixteen years ago many cars had bumpers equipped with isolators, whereas now most cars have foam core bumper systems. In the past many cars were constructed around a frame, today most cars are unibody construction. While most trucks retain the frame design, many vans are either unibody construction or a hybrid of the two. Although there have been advances in safety devices such as airbags and anti lock brakes, little has been done to protect the occupant in a rear impact[3].

The key to injury in a rear impact is typically energy. Based on the Laws of Physics, the energy into the collision must be accounted for in the elements of the collision. In most rear impacts, the energy into the collision is a function of the speed of the striking vehicle or the kinetic energy (KE) it possesses. This energy is partially transferred to the struck vehicle in the form of acceleration, partially dissipated in the components of the vehicles and partially retained as kinetic energy in the striking vehicle. The effect of each of these uses of the energy is necessary to understand the dynamics of the collision.

Mathematically, the principle can be represented by:

$$KE\ (V1)_{pre\text{-}impact} + KE\ (V2)_{pre\text{-}impact} = KE\ (V1)_{post\text{-}impact} + KE\ (V2)_{post\text{-}impact} + C_{rush}\ E_{energy}\ (V1) + C_{rush}\ E_{energy}\ (V2)$$

The limitation of the above equation is that there is no direct mathematical correlation between the damage to the striking vehicle, the damage to the struck vehicle and the kinetic energy transferred to the struck vehicle. Since crush energy is the visible damage and kinetic energy is the typical source of injury, the lack of a direct mathematical correlation indicates it is not possible to look at a vehicle and prove the accident could not injure the occupant. Similarly, the absence of property damage also does not establish the occupant was not injured. An indirect correlation does exist in that if there is visible damage to the vehicle, there was sufficient energy transferred to cause an injury. As discussed below, the absence or presence of an injury is a medical decision.

For reasons discussed in the biomechanics portion of this paper, it is common in a litigation setting for an expert to underreport the actual speeds of the vehicles. The reason for this error is the misapplication of physics principles in an attempt to imply a speed based on damage. The most common methodological error misapplies Newton's Third Law of Motion,[4] but the number of invalid models is extensive. For additional information, the reader can consult other papers including "Weakness of the Numerical Models Used in Accident Reconstruction Programs."[5]

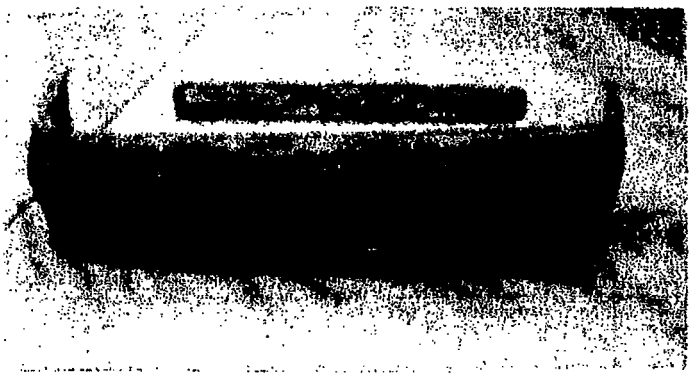

Figure 1. – Bumper cover with superficial damage.

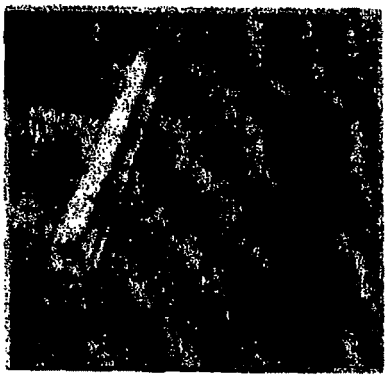

Figure 2. – Bent bumper reinforcement.

Caution that under ideal conditions, the use of vehicle damage can underestimate the speeds of vehicles by hundreds of percents.[6]

The constraints of the available data further compounds the limitation of looking at a vehicle to determine the energy transferred to the occupant. Among the areas of concern are the bumper system, the vehicle frame/unibody construction and the available photographs and estimates. An additional error source is the use of only one vehicle to determine speeds.

Bumpers. The bumper of a vehicle is not a safety device. As stated by the National Highway Traffic Safety Administration;

> A car bumper is designed to avoid or reduce damage in a low-speed collision. **It is not a safety device to prevent or reduce injuries to people in the car.** Rather, the bumper is designed to protect sheet metal parts of a car, as well as safety-related equipment such as parking lights and headlamps, in low speed collisions.[7]

Bumpers are designed to limit damage to the vehicle. The bumper system temporarily holds energy that could cause damage and releasing it in the form of kinetic energy before the damage occurs. However, this means that energy which could be absorbed by metal and plastic instead transfers in a fashion where it can injure occupants. Effectiveiy, the current design of bumpers increases the potential for injury to the occupant because less energy is absorbed in damaging the bumper.

There exists a common misconception that one can determine the damage to a bumper system by casual visual inspection. A foam core bumper system typically consists of three major components: the bumper cover, a foam core and a reinforcement bar. Figure 1 shows a bumper cover with apparent superficial damage. Figure 2 shows the damaged bumper reinforcement. Without disassembly of the bumper, the damage would not be apparent.

Frame/Unibody. A significant difference between unibody and frame construction is that the unibody transfers energy through more components than a frame does. The unibody construction results in the propagation of an energy pulse through the vehicle. A result of this, in rear impact collisions, is that the first visible damage may occur away from the impact site. It is not uncommon to see distortion to the roof or the hood in a rear impact. For this reason, merely concentrating an inspection on the rear of the vehicle may fail to identify all of the damage in a collision.

A common effect of the energy pulse is that an owner of a vehicle struck from

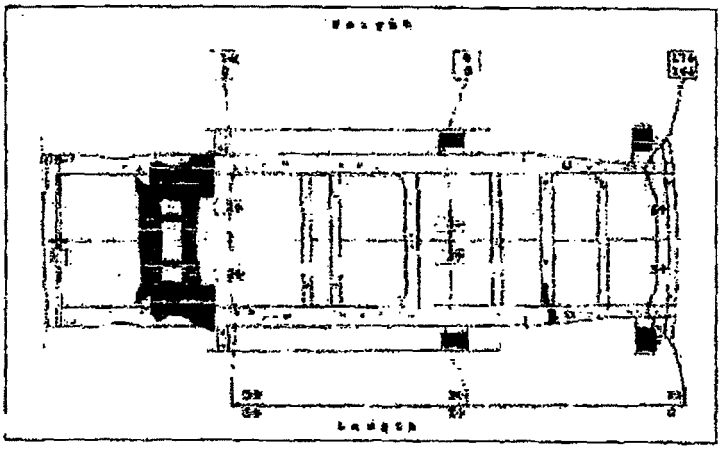

Figure 3. – Precision frame measurement.

Figure 4. – Vehicle with frame/unibody damage.

Figure 5. – Severe collision without significant damage.

behind may notice doors sticking, windows leaking, transmission problems, uneven tire wear and other problems not intuitively associated with the collision. Precision frame/unibody measurements can be obtained from many repair facilities to check for this type of damage. Figure 3 is an example of a precision frame measurement. In this instance, the vehicle has 17 mm of distortion from the rear impact that no repair estimate noted. Research indicates that damage to the unibody typically does not appear until impact speeds of 15 mph or greater.[8] Figure 4 shows a vehicle with significant unibody/frame damage that is not visible in the pictures.

A common error in litigation is to assert that the lack of visible damage to a vehicle implies a low speed. With the design of modern vehicles, in a bumper-to-bumper collision, it is rare to damage to the vehicles in collisions of under 10 mph. Numerous full-scale crash tests have been run at speeds up to, and exceeding 10 mph with no appreciable damage to the vehicles. As merely one example, a series of impacts in Texas with Ford Festivas resulted in no effective damage with vehicles involved in multiple collision up 11 mph.[9] Other full scale tests have also demonstrated this principle.[10]

Further compounding the problem is the significant quantity of energy that can be absorbed by vehicles before the onset of property damage. It is not uncommon for "no-damage" collisions to absorb 7,000, or more, foot-pounds of energy with no visible evidence. Many of the models used by experts in civil litigation routinely calculate energy values of zero to a few hundred foot pounds in collisions where there is damage, demonstrating the fallacy of their approach. Figure 5 shows a vehicle that was involved in a severe collision with effectively no damage. In this multi car collision, this vehicle caused several thousand dollars of damage to the car ahead of it which in turn caused hundreds of dollars of damage to the vehicle ahead of it.

Photographs and Repair Estimates

Another significant source of error is the use of repair estimates to determine velocities. Estimates available to the reconstructionist are often the preliminary estimate which is also known as the "estimate of record." If the vehicle is not repaired, the estimate will often miss damage that is not apparent in a visual inspection. For this reason, it is common for most preliminary estimates to carry a caveat noting that "hidden damage may be present" or a similar disclaimer. The relevant document for a repaired vehicle is the invoice listing the components actually fixed or replaced. Velocity determination based on repair estimates can provide minimum speeds only; actual speeds may be significantly higher.[11]

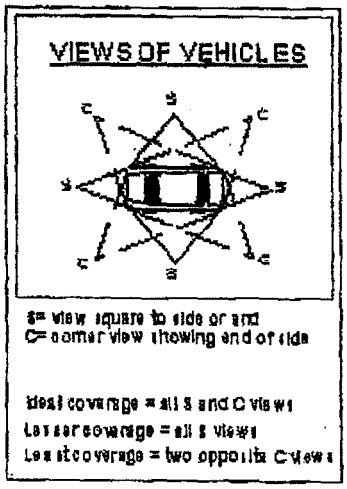

Figure 6. – Recommended views.

Limitations comparable to those found in repair estimates are present in the photographs commonly provided. In any collision, a thorough photographic record should include views of the entire vehicle.[12] Figure 6 shows the minimum set of photographs recommended - even in rear impacts - for several reasons including the issue of unibody distortion discussed above. In addition to these views, obtain close-ups of any damage.

A further limitation of photographs deals with the quality of the images pro-

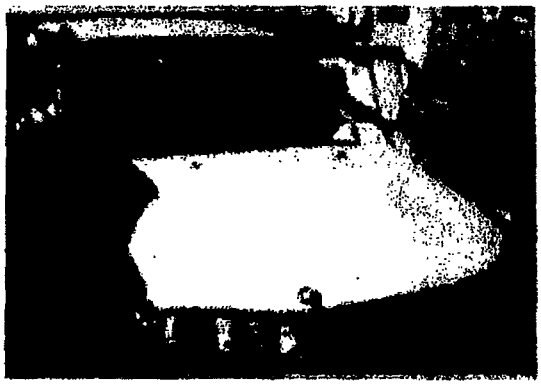

Figure 7. – Vehicle in storage.

Figure 8. – Improved imagery.

vided. Photographs often fail to capture the magnitude of the damage even when provided in color. Black and white versions of the photographs further exacerbate the issue. Figure 7 is a sample of the type of imagery the reconstructionist often receives.[13] Figure 8 is the same vehicle with the imagery taken in a more advantageous manner.

Figures 7 and 8 reveal the limitation of estimating speed based on photographs. While damage that is visible in photographs obviously exists, damage which is not visible in the photographs also often exists. Experts often demonstrate this by comparing the photographs supplied with the repair estimate. Figure 9 is a photograph of a vehicle with several thousand dollars of damage. Velocity estimates based on photographs can provide minimum speeds only; actual speeds may be significantly higher.

Use of one vehicle – It is not unusual for imagery to be available for only one vehicle in a collision. This may provide an incomplete understanding of the impact. Figure 10 shows the vehicle that struck the car shown in Figure 9. It is important to note that the initial collision between the cars was bumper to bumper[14]. The use of only one vehicle can provide minimum speeds only; actual speeds may be significantly higher.

The cumulative effect of these limitations is that in the majority of rear impact collisions, only minimum speeds can be determined. An expert who asserts they have determined the maximum speed typically does so by applying invalid methodology to insufficient data. It is worth noting that reasonable maximums can be determined. If there is actually no damage to either vehicle in a bumper-to-bumper collision, the relative impact speed[15] probably did not exceed 15 mph although documented cases of impacts in excess of 25 mph with no damage do exist. If there is minimal damage to the vehicles, a maximum relative impact speed of 20 to 25 mph is reasonable.

Depending on the goal of the analysis, the determination of impact speeds and changes in velocity may be irrelevant. The next section discusses this further.

It is appropriate at this point to address some other common misperceptions associated with the analysis of the speeds of vehicles.

Use of barrier crash test data is generally inappropriate. The damage in an actual collision is often directly compared to tests where the vehicle impacts a barrier. From the discussion of energy transfer above, the fallacy of this approach is obvious. In a barrier impact the energy is not used in accelerating the barrier or in damaging the barrier. The only place for significant amounts of energy to be absorbed is in damaging the vehicle. For this reason, vehicles without damage in 15 mph bumper-to-bumper collisions can have significant damage in a 5 mph barrier impact.

This explains why large vehicles fare so poorly in the IIHS[16] tests but do so well in an actual collision. The larger the vehicle, the greater the kinetic energy possessed for a given speed. The greater the kinetic energy in a barrier impact, the greater the damage. The empirical data available proves what the Laws of Physics lead one to expect.

The National Highway Traffic Safety Administration (NHTSA) sponsors numerous tests each year on motor vehicles. While most of these tests are barrier impacts, it is possible to find vehicle-to-vehicle crashes. Four tests were obtained using Honda Accords.[17] The vehicle-to-barrier test (VTB) was a frontal impact of a 1982 Honda Accord into a barrier at 34.8 mph. The three vehicle-to-vehicle tests (VTV) used 1984 Honda Accords. These are sisters/clones of the 1982 vehicle.[18]

Table 1 reveals that the average crush decreased in VTV impacts even when the kinetic energy of the vehicle heading into the crash increased by almost threefold. Be aware that there are too many possible impact variations to attempt a precise correlation between a staged test and an actual collision. However, the data in Table 1 does show that an attempt to assert that the damage from a

Figure 9. – Extensive hidden damage in a rear impact.

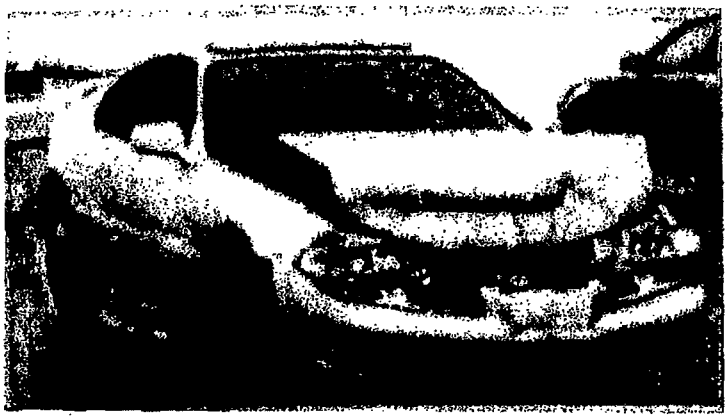

Figure 10. – Bullet vehicle.

Test Type	Closing Speed in mph	Average Crush in mm	% Average Crush	% Kinetic Energy
VTB	34.8	637	100	100
VTV	60.1	571	90	298
VTV	55.6	567	89	255
VTV	54.9	570	89	249

Table 1. – Damage comparison between VTB and VTV collisions.

barrier test can provide the velocity of the vehicles in car-to-car collisions has significant inherent error. The examples listed in Table 1 are at higher speeds than the authors of the paper implied they were dealing with. However, the design of bumpers is such that the effect of the protection provided by the bumper should be greater at lower speeds. This indicates a greater than four fold increase in energy is required to cause similar damage in the region commonly called "low speed."

Use of Insurance Institute for Highway Safety (IIHS) test data and repair costs underestimates the speeds of the vehicles. It is not uncommon to find individuals attempting to determine the impact speed of a vehicle by comparing the damage or the cost of repairs in an IIHS barrier impact. As demonstrated above, damage cannot be directly correlated. Additionally, the use of cost data for speed determination correlation is invalid. Even accounting for the effects of distortion due to variations in component costs, variations in regional costs throughout the country, variations in costs over time and variations in costs for non-OEM parts, the method still has unacceptably large error rates.

Small variations in impact configuration can result in significant variations in damage, and therefore cost, in collisions occurring at the same speed. Data from Neptune Engineering shows NHTSA tests at virtually identical speeds with significant crush variation. As an example, two tests on a Chevrolet Celebrity at the same speed had a 16% variation in crush depth.[19] Furthermore, two adjacent components can easily have a cost differential measured in thousands of percents.[20]

This leads to a subsequent problem in the approach used by some individuals to determine speeds. The source of the cost data is often IIHS, and it has been reported that the IIHS removes the bumper to check for damage. A damage estimator for an insurance company rarely does this. As discussed above, in many cases, damage is visible only after

the removal of the bumper system. For illustration, when the reinforcement and associated structures on a 2004 Ford Mustang are considered, the repair estimate for the vehicle would more than double when compared with the bumper cover alone.[21]

Biomechanical Response

Before discussing the effect of applied forces on the occupant in a rear impact case, it is useful to ensure the use of common terminology. Mechanics is the branch of physics that deals with the application of forces to an object. Bio means life indicating that biomechanics deals with the application of forces to something that is or was alive. Often biomechanics can be broken into three subspecialties. The first deals with prosthetic devices such as artificial limbs and organs. The second deals with sports and the physical actions of a body in motion such as enhancing the performance of a bicycle rider. The third area deals with traumatic events and the response of a person subject to trauma. This final category is the subject of this paper.

As applied to motor vehicle collisions and use outside of litigation, the biomechanics of trauma deals with how a given event injured a person. The most common application of this is to understand how an injury occurred in order to improve safety and develop methods to protect occupants. Examples include the development of seat belts, airbags, headrests, safety glass, etc. An application typically not found outside of litigation is the attempted use of biomechanics[22] to prove a person was not injured.

In a biomechanical analysis of a motor vehicle collision, there are two important questions. First, were forces applied where the diagnosed injury is located? Secondly, are the injuries reported of the types known to occur in a given class of collisions? As an example, and further discussed below, in a rear impact forces are applied to the cervical region of an occupant and cervical injuries are known to occur in rear impacts. Therefore, a cervical injury in a rear impact is biomechanically consistent with the applied forces. The fallacy of an injury threshold is discussed below.

As discussed above, the key to an induced injury caused by collision forces[23] is energy transferred to the occupant. If the struck vehicle moves in a rear impact, energy transfers to the occupant, forces apply and injury is possible. A common misrepresentation of the applied forces is the use of the misnomer "G-Forces." However, this approach fails to capture the complexity and severity of a rear impact collision. The key to injury in a rear impact is motion, both absolute and differential, not the specific peak or maximum acceleration. The myth of "G-Forces" and the associated canard of daily activities, are demonstrable in many ways, and many courts routinely disallow them. However, since they still appear, a brief discussion is warranted.

A rear impact subjects the occupant of the struck vehicle to hundreds of forces applied in a fraction of a second. These forces cause both absolute and differential motion of the spine and related structures. As an example, in a rear impact the cervical column undergoes a complex motion of both absolute and differential motion. The lower cervical column projects forward while the upper cervical column attempts to remain stationary. This results in the twisting the neck into an "S" shaped curve and the occurrence of differential motion between the vertebrae. It is this motion that is the injury mechanism, not the magnitude of one hypothetical "peak" acceleration. Figure 11 is an image of a cervical region twisted out of its normal lordosis by a rear impact. While it is theoretically possible that one of the hundreds of forces in a rear impact could match an applied force in a daily activity, there is no daily activity that subjects a person to the same myriad of applied forces in a fraction of a second.

An additional limitation of the use of the peak acceleration value is the difficulty in determining this value. Typically, the expert opining about peak acceleration is using a generic rule. Even under laboratory conditions where the many of the dozens of variables can be controlled, significant variations are possible for a particular speed. Freeman[24] showed variations exceeding 800 percent for the same velocity under controlled experimentation (Figure 12). Research has also demonstrated variations of 100 percent in a single individual tested under similar conditions. It is worth noting that the peak acceleration represents only one of the hundreds of forces on the occupant.

In a rear impact, the vehicle undergoes the resultant change in velocity in approximately 1/10 of a second. This applies regardless of the magnitude of the change in velocity until higher speeds are reached.

Since it is the movement of the vehicle that is the source of the injury energy, a reasonable question would be how much movement is necessary to cause injury. Looking at the structure of spinal columns reveals that localized movements of a fraction of an inch can lead to compromise of the spinal cord and even death. However, in reality a vehicle moving a fraction of an inch would not have that pronounced an effect on the occupant. Full scale testing has demonstrated that a movement of a few inches is biomechanically significant. If a vehicle propels forward even half a foot, expect significant occupant motion. This movement would occur with a change in velocity of 3 mph or less. This corresponds to symptoms being reported in published safety optimized test with changes in velocity of 2.5 mph[25] and in unpublished tests safety optimized tests with a change in velocity as low as 1 mph.[26] The issue of injury thresholds is further discussed below.

With an understanding that it is the rapid motion that causes the change in velocity, it is possible to look at the area

Figure 11. – "S" curve.

of injury and understand the reason for the damage. The following paragraphs discuss many of the common areas affected in rear impacts but do not address every injury.

Cervical. The presence of cervical injuries matches the applied forces that occur during the extension/flexion process. During a rear impact, the cervical region naturally pivots at C5-C6. In addition, the impact stresses adjacent areas to different degrees based on the induced rotation of the vehicle or the occupant. For this reason, it is not unusual to also find trauma to the adjacent C3-4, C4-5 and C6-7 regions. Various factors may also injure other portions of the cervical spine. In addition, a rear impact subjects the entire cervical column to tension, compression and shearing. Cervical injuries, including herniated discs, are associated with rear impacts.[27]

Lumbar/Sacroiliac. A rear impact subjects the lumbar region to a variety of forces. There is direct loading as well as differential loading from the seat back across the lordotic curve. Additionally, the torso moves upward during the ramping process. These forces subject the lumbar region to compression, tension and shearing and affect the entire lumbar region, but are higher in the area of L3-L4 due to the lordotic curve. If there is a rotational component to the collision, due to either occupant position or forces that do not pass through the center of mass of the vehicle, there are also angular forces applied. This situation alters the biomechanics of the collision and concentrates forces in the lower lumbar region to include the sacroiliac area. Lumbar injuries are associated with rear impacts.[28]

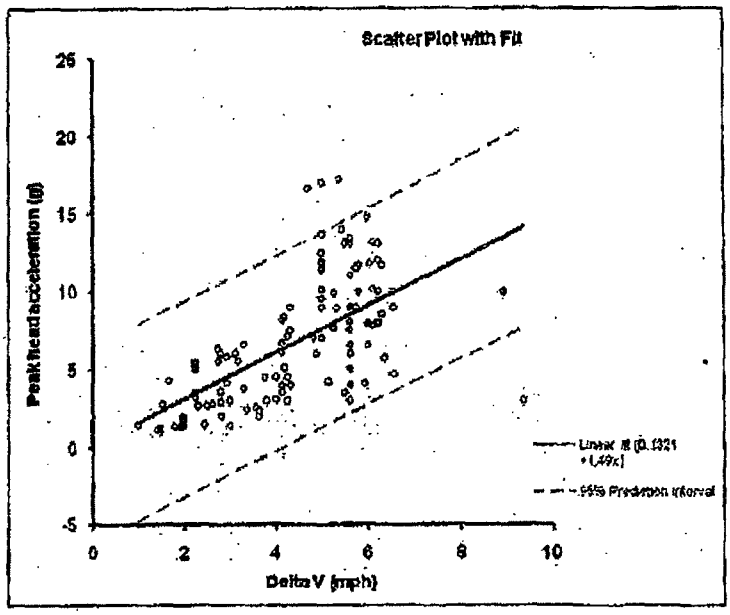

Figure 12. – Variations in peak acceleration.

Thoracic. In a rear impact, the thoracic region is subjected to a variety of forces. There is direct loading from the seat back as well as differential loading from the seat back across the kyphotic curve. Additionally, the torso moves upward during the ramping process. These forces subject the thoracic region to compression, tension and shearing. Thoracic injuries are associated with rear impacts.[29]

Brain/Head. Brain injuries are associated with five mechanisms in rear impacts. The first is direct contact with the headrest, the steering wheel or other portions of the interior of the vehicle. (The occupant may not remember this contact.) The second mechanism involves rotational forces applied during the extension/flexion process. The third involves shearing forces applied during the translation of the skull. The fourth identified mechanism deals with biochemical changes. The final mechanism is associated with vascular effects.

Shoulder. There are three primary mechanisms causing shoulder injuries in rear impacts. The first is direct contact from the seat belt or other portions of the interior of the vehicle such as the steering wheel. The second is differen-

tial loading of the shoulders and thoracic region due to the induced motion of the occupant. The third mechanism is load transference due to bracing on the part of the occupant. Any of these are capable of causing an injury to the shoulder.

Arms/Hands/Wrist. In a rear impact, a driver can apply voluntary muscles to grip the steering wheel. During the initial 100 to 200 milliseconds of the collision, the steering wheel is propelled forward ahead of the body. As the space between the steering wheel and the driver increases, the hands, wrists and arms of the driver are subjected to tension. After the initial movement, the torso of the driver moves forward and the hands, arms and wrists are subjected to compression. Depending on the dynamics of the particular collision, shearing may also occur. While less common, passengers who are projected forward towards the dashboard may also suffer injuries as they attempt to stop their motion. Injuries to the wrists of an occupant struck from behind are consistent with the applied forces.

Temporomandibular Joint (TMJ). The correlation between TMJ injuries and rear impact collisions is established. One possible injury mechanism involves the extension of the head/neck during the first 250 milliseconds of impact. The initial impact during a rear end collision leaves the head stationary and propels the torso forward. This puts the neck in tension. The tension produced by the neck pulls the lower jaw forward differentially with respect to the upper jaw. As full-scale tests reveal, the mouth opens during this part of the collision and the hinge point is the TMJ. Energy and motion cause injury to the TMJ. Full scale testing has also revealed an acceleration spike at the TMJ in rear impacts.

Who will be injured? Not every occupant is injured in every rear impact. Similarly, in a vehicle with multiple injured parties not every injured occupant will have the identical injury.[30] The reason for this is the extreme number of variables and permutations possible in a rear impact. These include, but are not limited to: gender, age, weight, height, neck length, neck circumference, cervical muscle tone, lumbar muscle tone, bumper design, impact angle, seat belt design, awareness, previous injuries, spinal degeneration, orientation of head, orientation of the neck, orientation of the torso, seat belt tightness, seat back design, seat back angle, etc. In calculations of the possible permutations, the value exceeds 1 sextillion. Compare this value with the few hundred total test subjects found in staged, safety optimized motion volunteer tests.[31] For this reason, it is not valid to attempt to infer injury potential from the volunteer studies.

While there is no numerical value for the injury potential of a given collision, numerous factors increase the likelihood of injury in a rear impact.

Occupant Position. An impact which induces occupant motion outside of straightforward and backward movement is more injurious. As the cervical, thoracic and lumbar regions move at an angle, there is a greater degree of stress to them. Angular acceleration or turning the body at the time of impact is an aggravating factor.[32]

Surprise. Published literature and basic engineering principles, reveal that an occupant who is struck by surprise is generally more likely to be injured than one who is braced.[33] Siegmund showed symptomatology at speeds lower than previously reported in staged, safety optimized tests simply by removing awareness of the precise moment of impact.[34]

Gender. Published research clearly reveals that women are more likely to be injured in traffic collisions than men.[35]

Predisposition to Injury. If a region has been previously damaged, injured or has degeneration, it requires less energy to damage the region again.[36] For example, potential preexisting changes to the spine would reduce the forces necessary to injure those regions.

Seatbelt Usage. The use of seat belts has been implicated in numerous injuries, including cervical and lumbar injuries.[37] The use of seat belts, as required by law, has the effect of decreasing the occurrence of fatal injuries. However, the principles of physics require the dissipation of energy. The seat belt concentrates the energy in the areas where it contacts the occupant. This results in an increased injury potential in those areas. Examples of areas stressed by seat belts are the lumbar region, the thoracic region and the shoulder. The seat belt also magnifies the motion in the cervical region and head, resulting in increased forces and increased injury potential on the neck and head, including the jaw.

Misconceptions. It is appropriate at this point to address some common misconceptions associated with the biomechanical response of the occupant.

It is not uncommon for it to be asserted that during a rear-end impact, the occupant of the target vehicle is propelled into his seatback as the vehicle accelerates forward. This is incorrect. The occupant obeys Newton's First Law of Motion[38] and remains stationary until acted upon by an outside force. The seat drives into the occupant. While the distinction may seem minor, it is critical to understanding the injury mechanics. This motion pushes the torso out from under the head and the cervical column undergoing extension. The neck remains in extension until the head overtakes the torso and then moves into flexion. The torso does not experience rebound until interaction with the seatbelt.[39]

The comparison of a rear impact to backing into an object is often promulgated and is incorrect on several levels. Backing into a wall is not biomechanically the same as a rear impact. When a vehicle backs into a wall, the occupant experiences extension and ride-down. There is very little cervical flexion. In a true rear impact, there is significant flexion. A simple analysis of the velocity curves of the vehicles in each case would show that there is no reasonable

reference frame where the curves are the same shape. In a rear impact, the vehicle accelerates significantly and then decelerates significantly. In a barrier impact, the vehicle decelerates significantly and then accelerates minimally. Figures 13 and 14 show the velocity profiles of a vehicle struck from behind and one that impacts a wall. It is obvious that the shapes of the curves are not the same.

Rear Impact Thresholds and Statistics

Threshold. It has never been established that there is a minimum speed change value below which people are not injured in real collisions. To the contrary, Professor Murray Mackay[40] has analyzed more than 2914 actual accidents reported in the U.S. National Accident Sampling System[41] and showed that there is no threshold speed change value for injury in real life (as opposed to staged) collisions. While Mackay concentrated on rear impacts, Kullgren and Kraft support the lack of injury threshold in rear impacts and demonstrated that there is also no injury threshold in frontal impacts.[42] Numerous other resources also support the lack of a threshold.[43]

Figure 15 shows the data from Murray Mackay.

Probability of Injury – The probability of injury does not exist. Freeman[44] has repeatedly discussed the epidemiology of injury. His work, and a basic understanding of probability, demonstrates why the retrospective analysis of the likelihood of an event has no meaning. By definition, once an event has occurred its probability of occurring is 100%. If there is only a 2% chance of injury of a person in a given event, once the person is actually injured, the probability becomes 100%.

Prospectively an attempt to determine the probability an injury will occur can be made. If a test were run with 100,000 people at different speeds and 90 percent were injured in an impact above a given velocity, a projection of the future risk could be made. However, it would not prove that the remaining 10 percent were not injured. The medical treatment of individuals by its nature concentrates on the injured occupants, not the entire set of possible occupants. If there is only a ten percent risk that a given population is injured, it is those ten percent who are expected to seek medical help.

Conclusion

The absence or presence of vehicle damage is not a reliable indicator of injury potential in rear impacts. Damage reports often only include superficial damage, and do not consider that the frame of the car or bumper may have been structurally compromised. Images of the damage may also be misleading, as they frequently are incomplete or of poor quality. Also, references for damage are often from faulty studies and assumptions, such as those which correlate damage from rear impacts with that of barrier impacts.

Based upon the principle of conservation of energy, any energy which does not go into damaging the vehicle must convert into kinetic energy, the source of injuries. Structures such as the bumper may protect the car from damage, but do not protect the occupants, who receive the excess kinetic energy. Also, consider that large amounts of energy may transfer to the occupants before the onset of damage to the vehicle.

Figure 13.

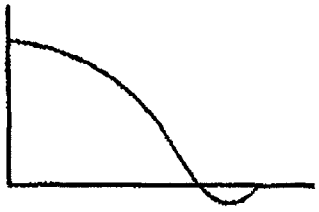

Figure 14.

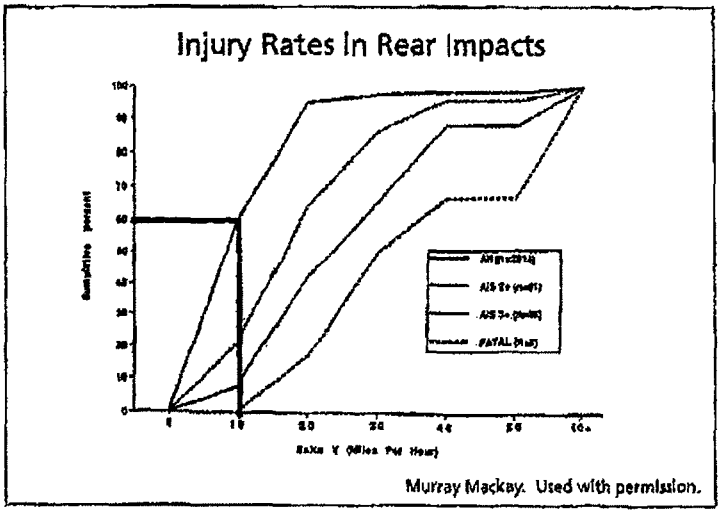

Figure 15. – NASS injury rates in rear impacts.

Furthermore, a particular set of injuries is common in rear impacts. Among these are injuries to the neck, head, and back resulting from the initial impact as well as injuries to other parts of the body during rebound. These injuries, while consistent with the type of impact, are dependent upon innumerable variables involving the occupant and impact specifics. As such, it is impossible to determine an injury threshold under which individuals will incur no injury.

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Endnotes

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11 It is not uncommon for a repaired vehicle to exhibit unrepaired unibody/frame damage that the body shop failed to note.

12 In some instances, it may not be physically possible to photograph the entire vehicle. This most commonly occurs in salvage yards where other vehicles block the access to the subject vehicle.

13 This vehicle was not involved in a rear impact but illustrates a principle. It is not uncommon for experts to opine on the amount of crush based on photographs such as this.

14 During mediation in this collision the insurance company provided pictures of the struck vehicle. When the plaintiff produced picture of the striking vehicle the defense dropped the "low speed" argument.

15 Relative impact speed is the difference between the velocities of the vehicles at the moment of impact. If the struck vehicle was stopped, the impact speed equals the relative impact speed.

16 Insurance Institute for Highway Safety, www.highwaysafety.org.

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21 *Id.*

22 Another term often used in litigation is biodynamics.

23 The startle or stretch response of an occupant, although relevant, is not a subject of this paper.

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1/03 - 4/04: Battalion Commander, US Army Corps of Engineers - Baghdad, Iraq, Kuwait City, Kuwait, Ft. Carson, CO

- Civil Engineering (Civilian and Military Infrastructure).
  - Water Distribution, Power Generation, Power Distribution, Roadway Repair/Improvement/Development, Irrigation Systems, Sewage & Sanitation Systems, School Renovation, etc.
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12/91 - 1/03: Engineer/President, Raymond P. Smith and Associates - Denver, Colorado

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Participated in engineering programs in the following areas:

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Participated in engineering programs in the following areas:

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12/81 - Present: U.S. Army Corps of Engineers - Current Rank of COL
Present Military experience includes the following areas:

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- Masters Degree - Strategic Studies, U.S. Army War College, Carlisle, PA 2008
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- U.S. Army Command and General Staff Officer Course
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- Ordinance Officer Branch Qualification Course

Professional Organizations:

- National Society of Professional Engineers
- Society of American Military Engineers
- IEEE - Engineering in Medicine and Biology Society, Remote Sensing Society
- Society of Automotive Engineers
- Accident Investigation and Reconstruction Practices Standards Committee
- Association for the Advancement of Automotive Medicine
- The Army Engineer Association
- Veterans of Foreign Wars
- American Legion
- Disabled American Veterans
- Member of the Editorial Board:Journal of Whiplash & Related Disorders

Continuing Education

- 1992 Stapp Car Crash Conference
- 1993 SAE Conference
- 1993 21st International Workshop on Human Subjects for Biomechanical Research
- 1993 Child Occupant Protection Symposium
- 1993 Stapp Car Crash Conference
- 1993 Head & Neck Injury Symposium (SAE)
- 1994 SAE Conference
- 1994 Rear Impact Symposium (SAE)
- 1994 22nd International Workshop on Human Subjects for Biomechanical Research
- 1994 Stapp Car Crash Conference
- 1995 SAE Conference
- 1995 23rd International Workshop on Human Subjects for Biomechanical Research
- 1995 Stapp Car Crash Conference
- 1995 Accidental Injury: Biomechanics & Prevention, University of California, San Diego, Office of Continuing Medical Education
- 1996 SAE Conference
- 1996 Biomechanics of Accidents, Texas A&M University System
- 1996 AIP Crash Tests
- 1996 AAAM Conference
- 1996 24th International Workshop on Human Subjects for Biomechanical Research
- 1996 Stapp Car Crash Conference
- 1996 Impact Head Injury; Responses, Mechanisms, Tolerance, Treatment & Countermeasures (NATO)
- 1997 SAE Conference
- 1997 Airbag Design and Performance TOPTEC (SAE)
- 1997 Perception-Reaction-Conspicuity Seminar, University of Iowa
- 1997 AAAM Conference
- 1997 Child Occupant Protection Second Symposium
- 1997 Stapp Car Crash Conference
- 1997 25th International Workshop on Human Subjects for Biomechanical Research
- 1998 SAE Conference

- 1998 Photogrammetry in Accident Reconstruction
- 1998 AAAM Conference
- 1998 26th International Workshop on Human Subjects for Biomechanical Research
- 1998 Stapp Car Crash Conference
- 1998 Whiplash Symposium
- 1999 SAE Conference
- 1999 Current Issues in Using Crash Injury Data
- 1999 Introduction to Biomechanical Trauma
- 1999 Ergonomics
- 1999 Applied Research Methods
- 1999 Directed Research
- 1999 Health Care Management and Administration
- 1999 27th International Workshop on Human Subjects for Biomechanical Research
- 1999 Stapp Car Crash Conference
- 1999 3rd Annual Crash Injury Research & Engineering Network Conference
- 1999 Legal and Ethical Aspects of Health Care Administration
- 1999 Biomechanics of Hard and Soft Tissue Injuries
- 1999 Directed Research II
- 2000 SAE Conference
- 2000 Biomechanics of Motor Vehicle Accidents
- 2000 Directed Research III
- 2000 Neurology I
- 2000 Orthopaedics and Rehabilitation of the Spine and Upper Extremities
- 2000 Biomechanics of the Spine and Upper Extremities
- 2000 Directed Research IV
- 2000 Neurology II
- 2000 Orthopaedics and Rehabilitation of the Lower Extremities and Sports Injuries
- 2000 Biomechanics of the Lower Extremities and Sports Injuries
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- 2001 29th International Workshop on Human Subjects for Biomechanical Research
- 2001 Stapp Car Crash Conference
- 2002 SAE Conference
- 2002 AAAM Conference
- 2002 30th International Workshop on Human Subjects for Biomechanical Research
- 2002 Stapp Car Crash Conference
- 2005 International Whiplash Trauma Congress
- 2005 SAE Conference
- 2006 International Whiplash Trauma Congress
- 2007 Composite Risk Management Basic Course
- 2007 Accident Avoidance Course for Army Motor Vehicle Drivers
- 2007 International Whiplash Trauma Congress
- 2008 SAE Conference
- 2009 SAE Conference
- 2010 SAE Conference
- 2010 Humvee Rollover Egress training
- 2011 SAE Conference

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- Countering the Opposition's Biomechanical Expert, June 15, 1998 Biomechanics of Motor Vehicle Accidents, Instructor Lynn University, Masters of Science in Biomechanical Trauma 2000
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- Accident Reconstruction and the Biomechanics of Head Injuries, August 9, 2002
- Biomechanics and Accident Reconstruction, November 7, 2002
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US States Where John Smith Has Been Recognized by Courts as an Expert

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- Georgia
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- Louisiana*
- Missouri
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- Oregon
- Texas
- South Dakota
- Virginia
- Wisconsin
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Publications of John Smith

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The Problem with Probability

How to spot when defense experts misuse probability in your auto case, and what to do about it.

by Michael D. Freeman

Probability forms a basis for many decisions people make. When you buy a new DVD player and opt not to purchase the extended warrantee for an additional $30, you are evaluating, consciously or subconsciously, a series of probabilities. Many factors-how much the unit costs, your prior experience with DVD player failure, and how long you usually keep electronics before replacing them with an updated model-affect your decision that it is less than likely that the $30 expenditure is justified.

Probability plays a pervasive, important, and typically hidden role in virtually every auto injury case. Both plaintiff and defense experts rely extensively on probability or risk (used synonymously in this article) to persuade a judge or jury that their opinions are valid. These opinions affect how the fact-finder perceives issues-such as causation, negligence, and injury severity and prognosis-that dictate trial outcomes.

Unsubstantiated probabilities are often used to bolster weak or completely unsupported expert testimony. Contrary to some trial lawyers' beliefs and practices, testimony that improperly relies on probability does not become more valid when offered on behalf of an injured plaintiff.

MIST v. MAID

The defense's use of probability in minor impact/soft tissue (MIST) cases differs from its use in major auto injury and death (MAID) cases in several ways. The defense uses probability after the fact to deny causality in MIST cases. For example, an expert will say it is within the realm of possibility that a plaintiff who developed neck pain within a day of a minor collision, or felt arm pain within a week, or was diagnosed with a herniated cervical disc within a month had these symptoms before the collision, was injured some other way, or is not really injured. This allows the defendant to ask the jury to speculate about some other, unnamed injury cause.

In contrast, the defense cannot claim that a plaintiff who has been catastrophically injured or killed in a high-speed collision was paralyzed or dead before the collision or was injured in some other way shortly afterward. In MAID cases, the defense must account for the injury while pointing to a higher probability that the failure to wear a seat belt or the plaintiff's excessive speed, for example-rather than a product failure or the negligence of the defendant-caused the injury.

In MIST cases, statements of probability form the entire basis for the defense strategy. The defense will tell the jury that injuries are *unlikely* when there is minimal damage to a vehicle. Defense experts will claim that at low speeds, injury is so *improbable* that it is virtually impossible. Defense medical examiners will opine that most patients will recover from injury in a matter of weeks or months, and so the plaintiff's protracted recovery is so unlikely that it must be due to some other injury or preexisting condition.

With these proclamations, the defense and its experts are telling the jury: If something is unlikely to have happened, then it *probably* didn't happen. Many judges will allow such testimony over the plaintiff's objections, relying on the jury to assign the appropriate weight to the opinions they hear.

The defense also relies heavily on probability in MAID cases, particularly those involving products liability claims. For example, in jurisdictions where defendants can argue that a plaintiff contributed to his or her injuries by not wearing a seat belt, they often claim that had the plaintiff worn a seat belt, injury would have been highly unlikely. So, they assert, the plaintiff's injuries were caused entirely (100 percent probability) by his or her failure to use a seat belt. The defense often uses expert testimony advancing this theory in rollover and ejection cases, and because it's well known that wearing a seat belt prevents occupant ejection, plaintiff lawyers rarely object to the testimony.

Another way the defense may use probability in MAID cases is by claiming that a failed safety device, such as a seat belt or air bag, did not contribute to the injury because the crash itself was so devastating. In other words, if the same crash occurred again and the device didn't fail, the plaintiff's injuries would be the same.

These are examples of "what if" scenarios, in which the expert essentially mentally reenacts the crash with different variables (for example, an air bag deploys rather than fails) and bases his or her opinion on the results. Courts typically scrutinize actual reenactments closely before allowing them to be shown to a jury, but these "thought experiments" are intended to show probable outcomes of imagined crash reenactments. There is no specified basis for the probabilities necessary to advance the opinion. There is no way to weigh the validity

of the claim, and the credibility given the testimony is based more on the credentials and presentation of the expert than the accuracy of the opinion.

The Solution

Plaintiff lawyers may be tempted to counter a defense expert's probability testimony by bringing in a consulting expert (for example, a crash reconstructionist, a biomechanist, or an engineer) to give an opinion on the likelihood that an injury would have occurred in a crash. But this approach is misguided. Some of the experts most willing to offer such testimony for the plaintiff also testify regularly for the defense that injuries are unlikely.

When both sides present such direct or rebuttal testimony, the result is a beauty contest between experts: the fact-finder is asked to choose the speculative testimony that he or she likes best. A better and more effective strategy is to attack defense experts' probability testimony by challenging the assumptions on which it is based.

Ultimately, all expert opinions must be rendered as "reasonable probabilities," an oft-repeated phrase that is poorly understood. Most experts understand that to be reasonably probable, an outcome must pass a threshold of being more than 50 percent of something, but that "something" is ill-defined. This lack of clarity can create problems.

For example, a defending attorney may challenge a plaintiff's expert who testifies about a rarely occurring injury, asking the expert to quantify its frequency. If the injury occurs only 1 percent of the time, the attorney may object to the testimony as speculative because the injury does not meet the 50 percent threshold for reasonable probability-a misapplication of the standard. On the other hand, some defense experts will testify to a reasonable certainty (defined as a 100 percent probability), as the term sounds more convincing than probability.

Most statements of probability must be supported by population-based data or epidemiologic studies to survive an evidentiary challenge, based on *Frye v. United States*[1] or *Daubert v. Merrell Dow Pharmaceuticals, Inc.*,[2] and other cases. Here are some simple rules of thumb to keep in mind.

For MIST cases:

- *Risk is a predictive tool, and you cannot predict the past.* Risk or probability cannot be used retrospectively to cast doubt on or deny an injury that has been observed and recorded. The defense may try to use the low likelihood of injury after a crash to deny that the crash caused the injury.

  A good measure of such statements' validity is to consider how they would sound if a death rather than an injury had resulted from a crash. Just as the low risk of death in a particular circumstance cannot be used as a basis for the jury to ignore the testimony of a pathologist, the low risk of injury cannot be used to ask the jury to ignore the testimony of a treating physician. Risk is exclusively a predictive tool: It can be used in theoretical or "what if" scenarios (for example, What if the crash had not involved a second impact?), but it cannot be used to deny an injury outcome as reflected in a medical record.

- *What is "usual," "normal," "typical," or "average" has no application to a specific case.* References to average injury responses to a crash are irrelevant to individual outcomes. The defense may use expressions like "most people" or "usually" when discussing a plaintiff's injuries in an effort to cast doubt on them because they are somehow out of the ordinary. Even if it is true that the average person would not suffer permanent injuries in a particular crash-a statement that would have to be validated with real data-this does not mean that 30 percent of the population would not be permanently injured, or that the plaintiff is "average." A good analogy is body weight: If the average person weighs 170 pounds, this does not mean that the next person who walks through the door cannot weigh 200 pounds.

- *Injury risk is population-specific.* Gender, age, physical condition, vehicle type, and other variables all contribute to injury risk in a crash. A 53-year-old woman with a history of neck surgery belongs to a relatively rare demographic group, and so the claim that injury from a minimal-damage crash is highly unlikely in the general population has little meaning for her case. Even if accurate, such claims are relevant only when there is no specific individual with an injury. Once the injury has occurred, the various risk factors for injury present in the individual define a target population for which injury-frequency statistics do not exist.

  For example, while it may not be worth arguing with a defense expert over the claimed frequency of

cervical disk herniation in the general population exposed to an under-10-mph crash, the plaintiff attorney can cross-examine the expert about the risk of disk herniation in the general population of 53-year-old women with prior neck surgery. The question is impossible to answer accurately beyond stating that the risk is greater than for the general population.

- *The opinion must have a sound basis.* When an expert testifies that injuries are unusual in crashes with less than a certain amount of vehicle damage, this opinion must be challenged. It implies knowledge of injury risk for such crashes, and risk is a population-based inference. Evidentiary standards, such as those established by *Frye and Daubert*, allow for hearings in which unfounded and speculative claims of probability can be challenged and excluded.

For MAID cases:

- *Again, the opinion must have a sound basis.* Defense claims regarding vehicle safety features, such as seat belts and how they might have affected a crash outcome if they had been used, must be supported with data to be quantified and validated. For example, seat belts are not designed to prevent injury. They are primarily designed to reduce the risk of ejection during a rollover. The degree to which they reduce that risk—and to which an ejection increases the injury rate—are matters of scientifically validated fact, not conjecture. While it is reasonable to state that failure to wear a seat belt increases ejection risk, it is unreasonable (and false) to say that a belted occupant is *never* ejected and that unbelted occupants are *always* ejected.

- *Comparisons must be appropriate to the case.* The defense often uses probability and statistics to obscure product defects on particular vehicles by demonstrating that the vehicle has similar or better fatal crash rates than others in its class. Since more than 95 percent of fatal crashes are the result of driver error, comparing the rates of all fatal crashes will hide real differences between vehicle types for crashes caused solely by a manufacturing defect.

- *Data must be applied properly.* The defense's use of risk often involves hidden or misleading denominators. For example, it may be correct that twice as many serious injuries occur in 10-mph frontal collisions when the occupants aren't wearing seat belts. However, if the opinion came from data showing that out of 100 such collisions (the denominator), there were only three serious injuries—one restrained and two unrestrained (the numerators)—then this data merely means that serious injuries are rare in such collisions regardless of restraint use.

Statistics can be used in almost any way imaginable to support an expert's opinion. Plaintiff lawyers must understand all the underlying parameters of a statistic to know when it is being applied properly or improperly.

Caveats

Be alert for cited studies for which the expert cannot specify the sample size, sample population characteristics, or study methods. Many experts hear about a study thirdhand or merely review an abstract before basing an otherwise unsupported opinion on it. Many publications designed to assist the defense of auto injury and death litigation incorrectly summarize articles or improperly extrapolate authors' conclusions.

Also be suspicious of any expert who cites 20 or more publications to support his or her opinion. In a recent case in the author's experience, the defense biomechanical expert cited 68 papers that she claimed supported her testimony. The author reviewed all 68 papers and found that not only did none support the opinion, but more than 50 had nothing to do with the type of crash involved in the case. Opposing counsel withdrew the expert when the plaintiff pointed this out.

When testifying to a reasonable probability, an expert is saying that he or she is more than 50 percent certain that the expressed opinion is accurate for the circumstances of the particular case—nothing more. Do not let your expert get trapped by opposing counsel's definition of what a reasonable probability implies.

By the same token, when an opposing expert testifies to a reasonable certainty, do not let the claim stand without determining the expert's rationale for making it. This is particularly true with testimony arising from the types of "thought experiments" described above, the results of which could hardly be called certain.

A sharp ear and eye will help lawyers identify potentially problematic and specious probability-based testimony early in the discovery process. Filing motions to exclude such testimony will allow fact-finders to make reasoned and unbiased determinations of negligence, causation, and damages. If the case and the opposing expert testimony warrant it, retain a consulting expert to reinforce the issues.

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Notes:

1. 293 F. 1013, 1014 (D.C. Cir. 1923).
2. 509 U.S. 579 (1993).

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Review

Applications and limitations of Forensic Biomechanics: A Bayesian perspective

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ABSTRACT

Forensic Biomechanics is an analytic method intended for presentation in a court of law. The method consists of the reconstruction of an injury mechanism followed by a comparison between the injury risk of the mechanism and the injury tolerance of the individual. In recent years some courts have excluded such testimony based, in part, on the inability of experts to quantify the potential error of the methods they relied upon in reaching their conclusions. The application of Bayes' Law to a forensic test of truth in a disputed matter allows for quantification of the error inherent in the method through the conditioning of the pre-test probability of the test outcome with the true and false positive rate of the test. The result of the calculation is the Error Odds (O_E) for the test, or the ratio of correct to incorrect tests.

We present an Error Odds analysis of seven previously published case studies in Forensic Biomechanics as an illustration of the utility of the O_E as a metric for admissibility of testimony in the courts, with a minimum Error Odds ratio of 10 proposed as a threshold. The results of our analysis yielded only 1 of 7 cases of applied Forensic Biomechanics that surpassed the threshold for admissible testimony of 10, with most the cases falling below an O_E of 3. The results of the present study suggest that the forensic application of biomechanics is potentially fraught with error. We suggest that an Error Odds analysis be incorporated in Forensic Biomechanics as part of the analysis as a form of quality control and as demonstrable evidence of the accuracy of the methodology.

1. Introduction

In their paper entitled "Forensic Injury Biomechanics," Hayes et al. describe a method for estimating injury probability as a means of establishing the "biomechanical plausibility" of an injury when there is a dispute over the cause or some other aspect of the injury.[1] These authors advocate a three step method of biomechanical injury causation primarily directed at traffic collision injuries that first reconstructs the injury event forces, then uses a computer model of an anthropometric test dummy (ATD) to recreate the occupant movement and forces, and finally assigns a Factor of Risk (Φ) that represents the ratio of the crash forces to a previously described injury tolerance metric. The results are used most often in civil legal settings to either explain how an injury occurred (when used for the plaintiff), assert that an injury was unlikely to have occurred (when used for the defense), or describe the effect on injury risk of a hypothetical situation, such as the use of a seat belt (for either plaintiff or defendant).

These authors propose an intriguing concept; a biomechanical test for truth in disputed matters. The proposal is new territory for applied biomechanics; prior use of biomechanical analysis of real world (vs. experimentally produced) injuries has been largely confined to traffic crashes, and associated with efforts by the National Highway Traffic Safety Administration (NHTSA) or the National Transportation Safety Board (NTSB) to analyze crash-related injuries as a means of explaining them, or evaluating the effect that a safety device like a seat belt or airbag may have had on the outcome.[2,3] The initial determination of injury causation made by clinicians for NHTSA and NTSB (i.e. that the injury resulted from the investigated collision) is not altered by the biomechanical investigation; it is explicated. This differs fundamentally from the system described by Hayes et al. which is proposed as a means of determining "whether" rather than "how", for example, a herniated disc in the neck could result from a low-speed rear-end collision, among other tasks.

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New approaches to causal determinations in a forensic setting are carefully scrutinized by the opposing side, and occasionally rejected by courts as unsound. Recent court decisions have called into question the applicability of biomechanics in forensic settings, with one judge writing that an expert's opinion in a traffic crash injury case "*should be excluded for the reason that there is no scientific or medical basis for the use of biomechanics, engineering or accident reconstruction as a means to determine whether or not an injury has occurred as a result of a crash; this type of data is generally used to explain injury in an experimental setting rather than to explain away injuries that have already occurred*".[4]

It is reasonable to investigate the accuracy of such a methodology, since Forensic Biomechanics uses indirect measures such as applied force to serve as a proxy for injury risk (a population-based parameter), which in turn is used to assess the probability of injury presence (an individual characteristic). Epidemiologic observational study of injury is the gold standard for injury risk assessment[5] and measures intended to serve as a construct for risk that don't directly measure injury occurrence should be adopted and applied with appropriate caution. Conversely, a new methodology should not be rejected out of hand solely because it is new.

In the present paper, we describe a systematic assessment of the accuracy of Forensic Biomechanics, that is, how often such an analysis would be expected to be correct vs. incorrect. As part of our analysis, we examine the context of Forensic Biomechanics; whether it is used to determine "how" or "whether" an injury resulted from a traumatic event, or if manipulation of a hypothetical variable such as seat belt use would have made a difference in the injury outcome. Finally, we discuss how the epidemiology of injury plays an important but often hidden or neglected role in Forensic Biomechanics.

2. Injury and causation

Injury differs from disease largely by how rapidly symptoms are developed after an injurious event or exposure. For example, a fall down the stairs that results in a broken wrist is an injury, but carpal tunnel syndrome resulting from repetitive use of a key board is a disease.[6] In the former, the symptoms are temporally attributable to the fall as they occur immediately afterwards, whereas in the latter the symptoms may start in the middle of the night after 6 months of high intensity computer use. The determination of causation for a wrist fracture following a fall down the stairs doesn't require special expertise or knowledge, but the relationship between carpal tunnel syndrome and repetitive use of the hands is one that is not as easily recognized. For this reason, issues of injury causation are ordinarily decided by clinicians following generally accepted methods of causal determination based primarily upon the history of the event as related by the injured patient. This interaction helps establish the nature of the event and the temporal relationship between the event and the onset of symptoms. Clinical causation depends largely on the truthfulness of the injured party, and it is not unreasonable to question historical attributions of injury to an event from which compensation for injury can be recovered. Conversely, it cannot be said that just because compensation can be recovered for an injury that an injured party is likely to lie about the cause of his injuries. Forensic biomechanical injury assessment does not occur in a vacuum; all litigated cases have medical evidence of injury and the determination by a licensed clinician that the injury resulted from the injury mechanism. The following two *a priori* assumptions are generally valid in all cases in which such an analysis is requested:

(1) The attributed injury mechanism, a traffic crash for example, has occurred. It is assumed that the preliminary work of investigating the claim for an overtly fraudulent act, such as staging a collision, has already been performed, as insurance companies have Special Investigation Units (SIU) that are dedicated to this type of investigation and do their work long before litigation experts in Forensic Biomechanics are called in.[7]

(2) The injuries diagnosed by a claimant's physicians are real. This assumption is based on the work that would already be done by an insurer's SIU or others in investigating and identifying overtly fraudulent medical practices before a forensic biomechanical assessment is requested by a plaintiff or defendant. In some injury litigation claims a claimant may be found to be malingering (faking) or exaggerating an injury by an adverse medical examiner but the dispute as to whether the injury diagnosis is valid cannot be augmented by a biomechanical analysis. The assessment relates only to the causal relationship between the injury mechanism and the injury, and is unrelated to diagnostic validity issues. For example, if an injury was found to be physically impossible in a collision by a biomechanical analysis, say a traumatic amputation of a limb in a low speed collision, it is the injury *cause* rather than the injury *diagnosis* that would be disputed (an amputated limb would be difficult to fake). This is not to say that a biomechanical assessment of injury risk that resulted in a low injury probability would not fit with an explanation of malingered injury complaints, just that the determination of injury risk is independent of the injury or malingering diagnosis.

3. Forensic Biomechanics and probability

The two preceding assumptions dictate that determinations of probability based upon biomechanical assessments of injury risk must be approached with some caution when they are applied in a forensic setting. If it is assumed that the injury event occurred and the injury is real then it is reasonable to ask if a determination of biomechanical injury risk is even relevant to a determination of causation. As an example of how biomechanical injury risk can be misleading when making absolute determinations of injury causation, Tencer et al. described a cohort of 20 seat belt restrained crash victims with femur fractures that resulted from relatively low collision forces.[8] All of the crashes had been reconstructed for speed and a biomechanical analysis of the injury forces was performed following a vehicle interior inspection. The authors selected the cases because, when compared with experimental studies of femur fracture tolerance, most occurred at force levels equating to a biomechanical risk of fracture of 10% or less (this would correspond with a Factor of Risk of injury, as described by Hayes et al., of 0.1. In fact, two cases of fracture occurred at 1% risk and one case at 0% risk (Φ of 0.01 and 0.00, respectively). The low risk did not translate to low probability of injury, however, as all 20 subjects were injured, an unexpected but undisputed outcome. As a technique that is intended to discriminate between injury and no injury based on injury probability assessment, forensic biomechanical injury evaluation would lead to the erroneous conclusion that all of the fractures were improbable or even impossible. On the other hand such an evaluation could help explain why an injury that was deemed unlikely had occurred (e.g. Tencer et al. attributed the injuries to muscle forces acting along with the crash forces). The preceding example illustrates one of the pitfalls of an injury evaluation methodology that does not consider actual evidence or observation of injury in arriving at a conclusion of whether the injury was more probable than not.

This last point raises another issue to consider when evaluating the various applications of forensic biomechanical assessment of injury risk. All forensic testimony is given as "more probable or

likely than not" or as a "reasonable probability;" relatively interchangeable terms that serve as a quantifiable threshold that must be exceeded before the testimony is admissible.[9] Thus the expert must be "more than 50% certain" that the opinion is correct. Using probabilistic language for such testimony is somewhat of a mischaracterization of an internal process of the expert, who has opined that he is more certain than not that his opinion is accurate or true, regardless of the methods used to arrive at the opinion. When the opinion is scientific rather than medical, what constitutes a reasonable probability may result from an analysis of data, often from epidemiologic study. For example, if an alleged act of malpractice that altered the stage or projected aggressivity of a cancer was found to result in an increase in the probability of death within 5 years of more than 50% (from 5% to 10% for example), the conclusion that the plaintiff's life had been shortened could be given as a reasonable probability. In assigning a threshold value for injury probability vs. injury improbability, Hayes et al. create criteria for injury that can lead to erroneous conclusions. For example, if one interprets a reasonable probability of injury to be a Factor of Risk assessment that is 0.5 or greater, the application of this threshold to the cohort of 20 crash-related femur fracture cases described earlier would indicate that 16 of the 20 fractures did not occur, as only four were found to have a risk of more than 0.5. Some of this difficulty in translation comes from the conversion of a continuous variable (injury risk, 0–1 range) into a dichotomous variable (injury not present $\leqslant$0.5, injury present >0.5), and some of it comes from potential limitations on the extrapolation of data that is used to assess biomechanical injury risk.

As an example of the limited utility of some experimental and observation data for application in forensic venues and the problems introduced by dichotomization of such data, Hayes et al. describe a study by Newman et al. of National Football League (NFL) players who sustained concussions (mild traumatic brain injury) during plays that were filmed from multiple angles allowing for a reconstruction of the forces of the injury event.[10] The logistic risk curve that was fit to the data is displayed in Fig. 1.[11] The authors use the data from this study to estimate a Factor of Risk of concussion for one of their traffic crash case studies.

The graph in Fig. 1 demonstrates calculated Head Impact Power for nine professional football players who were found to have sustained a concussion, in comparison with 15 players who were impacted and not injured. The 50% risk level equates to a HIP_m of 12.8, a value that divides most, but not all of the data into injured and not injured. It is worth noting that 2 of the 9 (22%) players' concussions occurred at values below the 50% threshold (HIP_m of 8.9 and 12.2). Likewise, one of the 15 uninjured players (7%) sustained a HIP_m of 19.7, equating to a greater than 0.90 concussion risk, with no apparent ill effects. As an illustration of difficulties with the dichotomization of continuous data, had the nine injured football players been diagnosed with a concussion following a fall while at work or in a traffic crash, a forensic biomechanical analysis of the injuries that used the 0.50 point on the risk curve as a cut off would have resulted in 1 in 5 having their claim of injury rejected as "improbable".

As another example that raises questions about dichotomization of continuous data, Hayes et al. refer to the Neck Injury Index (N_{ij}) several times as a basis for assessing the probability of injury of various severities to the cervical spine. The N_{ij} is a metric used for evaluating the performance of airbags relative to the loads they may produce in the cervical spine.[12] In Fig. 2, a graph depicts the risk of AIS 2 injuries (an Abbreviated Injury Scale) of the cervical spine by N_{ij} (a nondimensional metric). AIS 2 injuries are considered "moderate" in severity, and include disc injuries and fractures without spinal cord involvement.

The probability of an AIS 2 injury at an N_{ij} of 0.5 is 18.9%, meaning that the graph predicts that if 100 people are subjected to an N_{ij} of 0.5 approximately 19 will sustain an AIS 2 cervical spine injury. If the threshold of a 0.5 N_{ij} is used to determine the probability that a cervical disc injury resulted from a traffic crash in an individual the test will be wrong one time for every four times it is correct.

This error rate for the N_{ij} as a forensic test of injury causation given above assumes that the risk curve can be accurately applied to all occupants in all types of collision, an assumption that is not necessarily valid. The N_{ij} risk curve is derived from a risk curve originally derived from porcine neck testing.[13,14] The curve was then matched to injuries observed in real world non-rollover frontal crashes by extrapolating from the N_{ij} measured in male crash test dummies generated during frontal barrier crash testing conducted at 35 mph. All of the crash tests involved airbag deployments and belted dummies. Although scaling and extrapolation can be performed to some extent, because the N_{ij} has not been validated for injury mechanisms outside of frontal traffic collisions any use of the metric for other injury mechanisms should be approached with caution.

There is certainly a justification for the development and application of new methods of evaluating the truth of assertions or allegations in disputed matters. A plaintiff in a civil litigation may have a completely legitimate claim of injury, but it cannot be denied that there exists a financial incentive to exaggerate or fabricate injuries and that exaggeration and fabrication occurs in some cases. Likewise, a defendant may have a legitimate reason for denying compensation to a claimant, but it must be acknowledged that there is a financial benefit to a defendant who prevents a legitimately injured plaintiff from being awarded compensation, and that in some cases claimants with real injuries are deprived of compensation. The fact finders' dilemma is how much weight they should give to the testimony of plaintiffs regarding their injuries, or to their treating physicians who may only be relying upon the plaintiff's history for a causal determination. A test that is intended as an impartial determinant of the truth in a disputed matter is an

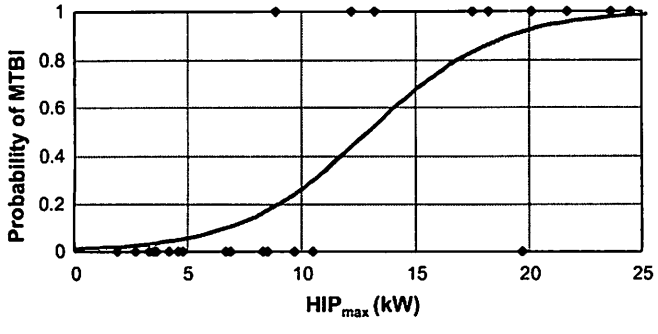

Fig. 1. Probability of concussion based on incidences of Mild Traumatic Brain Injury (MTBI) associated with the calculation for maximum Head Impact Power (HIPmax) (adapted from Newman et al.[11]).

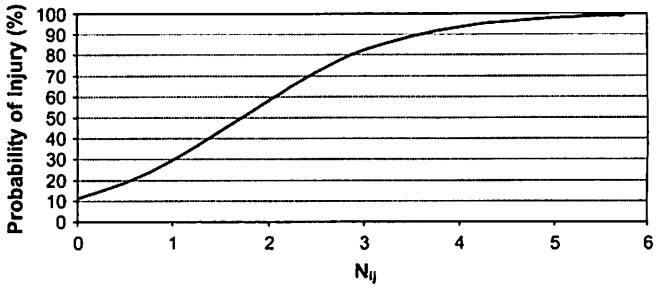

Fig. 2. Probability of an AIS 2 injury as predicted by the Neck Injury Index (N_{ij}) (adapted from Eppinger et al.[12]).

ideal solution for the fact finder only if the test can be shown to be accurate.

3.1. Assessing the error rate of forensic tests

In 1993, the United States Supreme Court issued an opinion in a case called *Daubert v. Merrell Dow Pharmaceuticals Inc.* The opinion set new standards for evidentiary hearings in the United States, in which the judge acts as a gatekeeper for proposed scientific testimony.[15] The Daubert decision laid out certain criteria for the admissibility of testimony that was based on new or novel methods, including four general standards considered by the Court to represent the basis for a valid scientific method, including:

(1) Whether the technique had undergone empirical testing; the theory or technique must be shown to be falsifiable, refutable, and testable;
(2) that the technique had been subjected to peer review and publication;
(3) that there is a known or potential error rate of the method; and
(4) whether or not the method is generally accepted by a relevant scientific community.

The Daubert standard has been used to challenge the admissibility of forensic biomechanical testimony on a number of occasions, and such testimony has been precluded as lacking in a scientifically sound basis on dozens of occasions in recent lower[16–22] and appellate court decisions[23–26], often because the expert cannot quantify the degree of error inherent in the analysis.

The error rate of a method, the 3rd listed criteria for an acceptable methodology under Daubert, is a good means of quantifying the accuracy of a method that serves as a test for truth in a disputed matter. When evaluating test accuracy, it is helpful to first understand how a test may be inaccurate. If a test is like a rifle with a scope mounted, then an accurate test would yield results like the target in Fig. 3a, in which the shooter knows that when the bulls eye is in the crosshairs of the scope that the bullet will strike the bulls eye. If there is too much random error in the test, in the rifle analogy this might be a scope that is mounted too loosely to the rifle, then the results will be unpredictable, like in Fig. 3b. If there is systematic bias in a test, if the scope is mounted to the rifle at an angle for example, then the results will be predictably wrong in a certain direction, as depicted in Fig. 3c.

3.2. Conditional probabilities and Bayes' Law

The purpose of any forensic test is to "condition" the probability of a particular outcome or result. For example, if the issue of interest is the probability of a particular injury "*A*" following a traffic crash, depicted symbolically as $P(A)$, then a conditioned probability would be the probability of injury given the presence of another factor; a positive test "*B*" for example. This conditional probability is depicted symbolically as $P(A|B)$; the probability of injury *A* given the positive test result *B*. An error that may occur when evaluating a conditional probability is that the assumption is made that the terms are reversible; that $P(A|B) = P(B|A)$. This error is called a Conditional Probability Fallacy.[27] An example of the Conditional Probability Fallacy is the conclusion that "90% of Spaniards speak Spanish" is equally true as "90% of Spanish speakers are Spaniards". As applied to a forensic test, the Conditional Probability Fallacy occurs when it is erroneously concluded that the probability that a test will be positive when a condition is present is the same as the probability that a positive test means the condition is present (symbolically represented as $P(\text{test positive}|\text{condition}) = P(\text{condition}|\text{test positive})$. As an example, one could devise a test for guilt that was based on body temperature; if the body temperature was above 50 °F the test would be positive for guilt. The test would be positive 100% of the time that the defendant was guilty, but quite obviously the defendant would not be guilty 100% of the time the test was positive. This type of error is also known as the Prosecutors Fallacy.[28]

The way to avoid a Conditional Probability Fallacy is through the application of Bayes' Law, the principles of which are critical to the evaluation of the potential error rate of a forensic test. Most simply stated, Bayes' Law allows for a more precise quantification of the uncertainty in a given probability. As applied in a forensic setting, Bayes' Law is a method of finding out what we want to know given what we know.[29] Bayes' Law is named for the essay by Reverend Thomas Bayes (1702–1761) on the statistical analysis of probability[30], and over the past 250 years subsequent authors have further defined and refined Bayes' original propositions. In more recent years Bayes' Law has been used in forensic settings primarily to quantify probabilities associated with DNA testing, however its use has expanded to virtually any disputed issue that requires the quantification of probability and its complement, uncertainty (uncertainty = [1 − probability]).[31,32]

At its most simple, Bayes' Law is stated $P(A|B)$ in which the probability of *A* is dependent upon condition *B*, vs. the probability of *A* absent any conditions. For example, the probability of drawing an ace of hearts from a deck of cards is 1:52; however the probability of drawing an ace of hearts given that only red cards can be selected is 1:26, the probability of drawing the ace of hearts when only hearts are drawn is 1:13, and the probability of the ace of hearts when only aces are drawn is 1:4.

When used to evaluate the accuracy of a test the application of Bayes' Law allows for the consideration of the most important conditions that can influence the ability of the test to arrive at a correct answer. As an example, we can hypothesize a test for the presence of illegal drugs that has a sensitivity rate of 0.90 (it correctly identifies test subjects with drugs in their system as positive 90% of the time) and a specificity rate of 0.80 (it correctly identifies test subjects with no drugs as negative 80% of the time). The sensitivity rate is the same as the *true positive* rate (0.90), and the complement

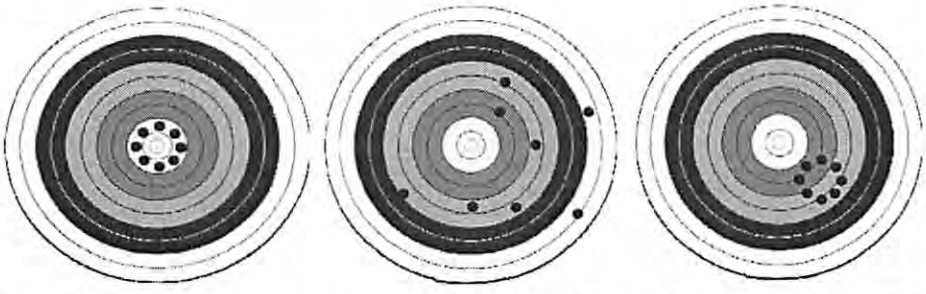

Fig. 3. 'Accurate' test results (a, left bullseye); 'Inaccurate' test results due to random error (b, middle bullseye); 'Inaccurate' test results due to bias (c, right bullseye).

of the specificity rate (1 – specificity) is the *false positive* rate (1 – 0.80 = 0.20). The ratio of the true positive rate to the false positive rate is also known as the Likelihood ratio for the test:

$$\text{Likelihood ratio} = \frac{\text{true positive rate}}{\text{false positive rate}}$$

3.3. Error Odds

In this paper, we introduce a simple application of Bayes' Law for assessing the degree of uncertainty in a positive test result, called the Error Odds (notated as O_E). In Bayesian terminology the Error Odds assessment is also known as the post-test or posterior odds. The result of the Error Odds assessment is the ratio of true positive to false positive tests given the expected "base rate" or frequency of the condition of interest in the population like the test subject (also known as prevalence). The equation for the Error Odds is as follows:

$$O_E = \frac{\text{true positive rate}}{\text{false positive rate}} \times \frac{\text{pre-test probability}}{(1 - \text{pre-test probability})}$$

The Error Odds assessment is unique in that it is designed for application in a forensic setting, and as such is only applicable to positive test results (true and false negative rates are not considered with the Error Odds). The rationale for a test validity measure that only considers true or false positives arises from the fact that the results of all forensic tests are ultimately presented as positive outcomes that support one or the other side of a disputed issue, regardless of the test outcome. As an illustration, a blood alcohol test administered in a drunk driving investigation that indicated the presence of alcohol would be presented by the prosecution to a jury as a positive test for guilt. The same test, if negative for alcohol presence, would be presented to a jury as a positive test for innocence. Thus the Error Odds assessment of a positive test result can be briefly described as the ratio of the rate of correct positive results to the rate of incorrect positive results. The Error Odds is intended as a "snapshot" of the degree of uncertainty in a test result. Note that the results of Error Odds calculation is actually the odds *against* error; this reversal is necessary so that a threshold for an acceptable level of error is a whole number below which the odds of error can be considered to be unacceptably high for application in a forensic venue.

As an illustration of the application of the Error Odds the results of drug testing of two hypothetical populations can be assessed for comparative validity. For the example, the true and false positive rates of the previously described drug test can be used (0.90 and 0.20, respectively). In order to complete the assessment a final piece of information is needed, and this is the prevalence or pre-test probability of drugs in the tested populations. The hypothetical test populations are: (1) a group of 100 prisoners recently incarcerated for a drug related crime in which drugs have been historically found in 95 of the subjects (0.95 drug prevalence) and (2) a group of 100 6th grade students in which drugs have been previously found in only 5 of the subjects (0.05 drug prevalence). Using the 0.9 true positive and 0.2 false positive rates the Error Odds assessment for the two populations is presented diagrammatically and in equation form in Figs. 4 and 5.

The calculation yields Error Odds of 86 and 0.24, or 86 correct test results for every 1 incorrect result for the prisoners and 0.24 correct results for every 1 incorrect result for the 6th graders. The Error Odds would be the same for any single individual test result. Note how sensitive the results of the accuracy test are to the prevalence of the condition of interest; even though drug presence is only 19 times more prevalent among prisoners than among 6th graders (0.95/0.05), the test is correct 358 times more often among prisoners than among 6th graders (86/0.24).

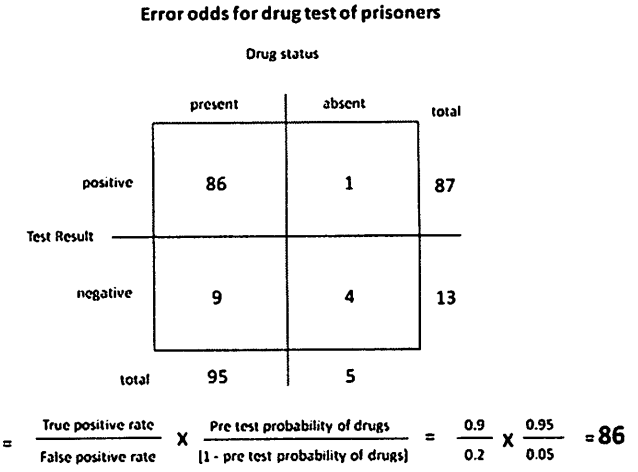

Fig. 4. A 2 × 2 contingency table summarizing the example analysis of the Error Odds associated with drug use in a hypothetical prison population.

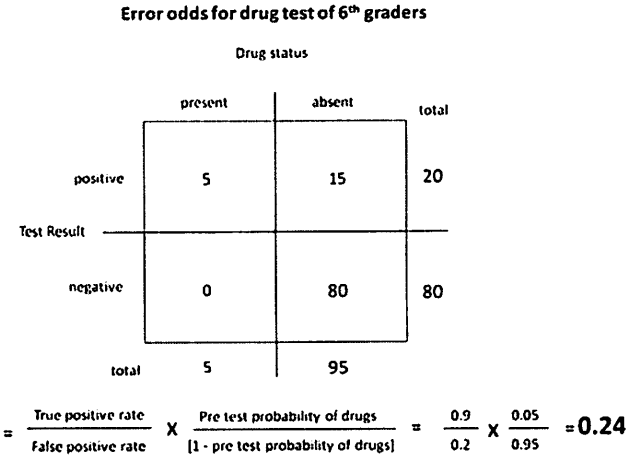

Fig. 5. A companion 2 × 2 contingency table supporting the example analysis of the Error Odds associated with drug use in a hypothetical elementary school population.

Also note in the example how the application of Bayes' Law avoided a Conditional Probability Fallacy or Prosecutor's Fallacy. The probability that the test would be positive given the presence of drugs was 0.9 for both groups however the conditioned probability that drugs would be present given a positive test was widely disparate between the two populations.

It is difficult to set an Error Odds value that qualifies a test result as "acceptable" for forensic testimony. It has been suggested that when Likelihood ratios are used to evaluate medical diagnostic test results a value of 10 is a minimum threshold from which to conclude that a patient has the condition for which he was tested.[33] In a similar vein, a lower threshold of 10 correct tests for every one incorrect test has been previously proposed for the evaluation of evidence in criminal matters.[34] With reference to a threshold ratio of 10, the matrix presented in Table 1 illustrates the Error Odds for a hypothetical forensic test with a true positive rate of 100%, given prevalence and false positive rates ranging from 0.05 to 0.95. The bolded cells on the right side of Table 1 are those that exceed 10. Note that if the pre-test probability of the condition is less than 0.5 the Error Odds do not exceed 10, regardless of the false positive rate.

Table 1
The breadth of Error Odds values assuming a true positive rate of 100%.

False positive rate	Pre-test probability						
	0.05	0.1	0.25	0.5	0.75	0.9	0.95
0.05	1.1	2.2	6.7	**20**	**60**	**180**	**380**
0.1	0.5	1.1	3.3	**10**	**30**	**90**	**190**
0.25	0.2	0.4	1.3	4	**12**	**36**	**76**
0.5	0.1	0.2	0.7	2	6	**18**	**38**
0.75	0.07	0.1	0.4	1.3	4	**12**	**25**
0.9	0.06	0.1	0.4	1.1	3	**10**	**21**
0.95	0.06	0.1	0.4	1.1	3	9	**20**

3.4. Error Odds modifiers

The presence of either bias and/or random error may increase the probability that a forensic test will yield an erroneous result by limiting the extrapolability of the data to the circumstances of the disputed matter, but the magnitude of effect may be difficult to estimate. Forensic Biomechanics relies in large part on experimental studies of animal, cadaver, and occasionally human volunteer subjects. Real world human tolerance levels demonstrate far greater variance than what can be produced experimentally for the simple reason that experiments cannot reproduce the breadth of variability inherent in the population, nor can they reproduce the variability of circumstances in which injuries occur. Fig. 6 illustrates a series of hypothetical Gaussian distributions that demonstrate the variability and quantity of experimentally derived data vs. the extent of variability in the general population. Error results when the experimental data is used as an index of risk for what may be observed in the general population.

The publication that forms the basis for the human tolerance metrics used in the Federal Motor Vehicle Safety Standards for automotive design and testing by the US Department of Transportation characterizes the extrapolability of human tolerance data to real world settings as follows[35]:

> "Such [tolerance] specifications are beyond the state-of-the-art in biomechanics except perhaps for a few academic situations. There are several difficulties which prevent a ready establishment of human tolerance levels. First, there are differences in judgment as to the specific degree of injury severity that should serve as the tolerance level. Second, large differences exist in the tolerances of difference individuals. It is not unusual for bone fracture tests on a sample of adult cadavers to show a three-to-one load variation. Presumably, variations of at least this magnitude exist in the living population. Finally, most tolerance levels are sensitive to modest changes in the direction, shape, and stiffness of the loading source. The above considerations indicate that complete and precise definitions of human tolerance levels will require large amounts of data based on controlled statistical samples. Only in this way can the influence of age, size, sex, and weight be comprehensively assessed and only in this way can mean loads and statistical measures of scatter be linked to specific tolerance levels."

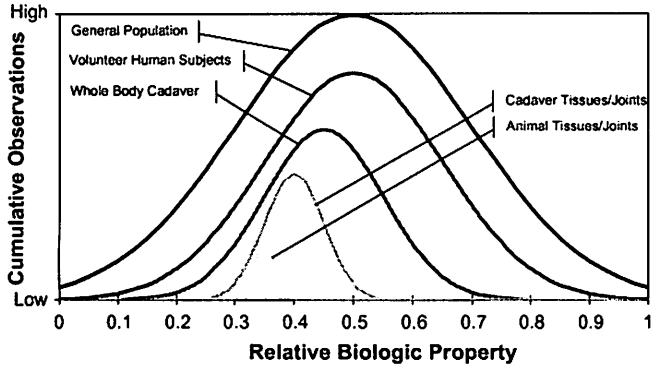

Fig. 6. A set of hypothetical Gaussian distributions demonstrating the variance, mean, and source of experimentally derived sample data compared with the general population.

The caution advised in the preceding statement refers to the effect of both bias and scatter on the extrapolation of tolerance specifications to real world occurrences as boundary conditions. We do not interpret this to mean that extrapolation of tolerance data cannot be accomplished in a forensic setting; rather, limitations and error should be identified prior to determining whether such data are suitable as a basis for a forensic opinion. This means that the influence of bias and random error should be evaluated in the methods and data used to arrive at the opinion.

3.5. Bias

When extrapolating from a study population to an individual in a Forensic setting it is ideal that the reference population be identical to the individual in every possible way in order to minimize bias. As this situation is a rarity, the next best approach is to characterize and quantify how the study population differs from the individual in aid of a determination as to whether the extrapolation is feasible. As an example, the study mentioned earlier by Newman et al. describing the injury mechanisms by which NFL football players sustained concussions raises the obvious question of whether the study population is more resistant to injury than the average motor vehicle occupant. The result is that values at which injury occurred in the study population are inflated, relative to the average motor vehicle occupant. The difficulty arises in assessing this potential difference; it would be a relatively simple task to quantify non-injurious proxies of injury resistance such as muscle strength in NFL players vs. the average motor vehicle occupant, however the degree of force required to cause a concussion is a different matter that may or may not be related to physical hardiness. It is reasonable to conclude however, that the extrapolation of data from a study group that consists of the most elite and physically hardy members of the population to an ordinary individual cannot occur without some degree of scaling, and that the failure to do so increases the probability of erroneous conclusions.

3.6. Random error

Lack of sufficient study numbers is the primary cause of random error, which only becomes an issue when the data are used as an exclusive rather than inclusive boundary condition. Referring back to the Newman et al. study on head injury, for example, the authors observed concussions at HIP_m levels as low as 8.9 kW. A reasonable conclusion from these data is that a concussion observed in any individual at a HIP_m of 8.9 kW or greater is consistent with the study data. The converse of this conclusion, that a concussion observed at a HIP_m of *less* than 8.9 kW is *not* consistent with the study data, would be erroneous to the extent that the limited number of injured subjects (here nine players) is insufficient to draw a sharply delineated boundary below which it can be said that injury cannot occur. The following analogy helps explain how the type of opinion (inclusive vs. exclusive) dictates the sensitivity of a test result to study numbers. If one had a box full of 100 balls that contained an unknown quantity of either red and/or black balls, and the balls could only be examined one at a time, if a single red ball was drawn one could conclude that red balls are present in the box at some unknown frequency. This would be a reasonable *inclusive* conclusion based on the limited sample. It

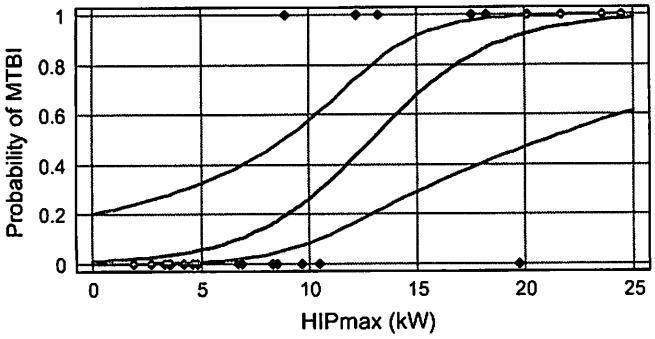

Fig. 7. Similar to Fig. 1, a 95% Confidence Interval (the red lines above and below the middle blue line) have been added to demonstrate the variability of MTBI associated with HIPmax. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

would be an error to conclude that black balls are impossible, however. This would be an *exclusive* conclusion that is only supported by the data if one ignores the effect of random error, which dictates that the sampled red ball may have been the only one in the box, and that rest of the balls in the box may be black. Only by increasing the sample size can one decrease the effect of random error.

One way to account for the effect of study numbers on random error (but not bias) is to present measures of central tendency such as a mean and regression with confidence intervals that take into account the variation among the data. As applied to the previously presented Newman et al. data[11] the 95% confidence interval for a 50% risk of concussion or mild traumatic brain injury (MTBI) among NFL football players would range from approximately 9 kW to 21 kW, as demonstrated in Fig. 7.

4. Case studies in Forensic Biomechanics; an Error Odds analysis

Hayes et al. describe their application of Forensic Biomechanics to three cases in which injury resulted from a traffic crash and four cases in which injury resulted from a fall. While it is estimated by the US Centers for Disease Control that there are more than twice as many fall-related injuries evaluated and treated in hospital emergency departments than traffic crash-caused injuries,[36] a much smaller proportion of fall injuries are litigated relative to traffic crash injuries. Taking into account the frequency of injury vs. the frequency of litigation the rate of crash injury litigation is estimated to be more than eight times greater than for falls,[37] and thus it would be expected that forensic biomechanical analysis of injury risk would be encountered significantly more frequently in traffic crash litigation than in slip and/or fall litigation.

4.1. Estimating Error Odds input values for various types of forensic biomechanical opinions

In the following section we present an Error Odds assessment of the seven case studies described by Hayes et al. as a means of demonstrating the utility of the methodology. This section is organized by the type of forensic biomechanical conclusions associated with each case. The cases were separated into three categories, relative to how the medical evidence of injury fit with the reconstructed circumstances of the injury mechanism. There are four cases (3 crashes and 1 fall) in which the major conclusions were deemed to be *explanatory*, in that they provided an explanation for how an injury occurred when the injury mechanism was not apparent (such as with an unobserved fatal fall). There are two cases (1 crash and 1 fall) that included an *alternate hypothetical* scenario in which the theoretical effect of an injury reduction mechanism such as a seat belt was evaluated and compared to the actual event. The third category of conclusions, *refutative*, is illustrated with a traffic crash case in which a diagnosed injury was deemed unlikely to have resulted from the collision.

This section is organized as follows; first, a synopsis of the case details from the Hayes et al. paper is presented. This is followed by estimates of the three elements needed for the Error Odds assessment; the pre-test probability and the true and false positive rates of the test. Values are reported in ranges when appropriate, and estimates have been adjusted to favor a lower error rate in order to maximize the Error Odds value. Estimates of the effect of bias and error on the values are also presented when warranted. The results of the Error Odds calculations for all of the cases are presented in Table 2.

The most difficult value to estimate for the Error Odds analysis was the pre-test probability of the condition of interest. This value is not the same as simply determining the prevalence of the injury given the circumstances, however. A Bayesian (conditioned) perspective is helpful to understand the pre-test probability necessary for an Error Odds assessment. As an illustration, if 100 occupants are exposed to a 10 mph collision, there may be only 20 who are injured, resulting in an injury prevalence of 0.20. If all of the 20 injured occupants made a claim for injury then the prevalence of injury among the claimants would be 1. If 10 of the 80 uninjured occupants made a false claim for injury in addition to the 20 injured occupants then the frequency of real injury among all claimants would be 20 out of 30, resulting in a pre-test probability of 0.67. It is this value that would be used for an Error Odds assessment, rather than the 0.20 or 1, both of which would result in a Conditional Probability Fallacy.

Thus, in the proceeding analysis section, the pre-test probabilities for some of the Explanatory and Refutative cases were arrived at not by asking "*how often do these injuries occur*" but rather "*how*

Table 2
Input values supporting Error Odds calculations for the re-examined case studies.

Analysis type	Case no.	Description	Pre-test probability (PTP)	True positive (TP) rate	False positive (FP) rate	Likelihood ratio (TP/FP)	Pre-test odds (PTP/[1 − PTP])	Error Odds (O_E)
Explanatory	1	Traffic crash neck injury	0.93	0.5 (Any injury) 0.5 (long term)	0.5 (Any injury) 0.5 (long term)	0.5 (Any injury) 0.5 (long term)	13.3	**18.5 (Any injury) 30.2 (long term)**
	2	Slip and fall in ramp	0.5	0.89	0.64	1.39	1	**1.4**
	3	Fall from balcony	0.35	0.35	0.65	0.54	0.54	**0.3**
	4	Fall on trampoline	0.53	0.53	0.38	1.39	1.13	**1.6**
Hypothetical	5	*Traffic crash belted vs. unbelted*	*0.8 (Head) 0.53 (neck)*	*0.8 (Head) 0.53 (neck)*	*0.8 (Head) 0.53 (neck)*	*0.8 (Head) 0.53 (neck)*	*0.8 (Head) 0.53 (neck)*	**16 (Head) 2.4 (neck)**
	6	*Exercise bike fall no safety vs. safety*	*0.99 (No safety) 0.5 (safety)*	*0.99 (No safety) 0.5 (safety)*	*0.99 (No safety) 0.5 (safety)*	*0.99 (No safety) 0.5 (safety)*	*0.99 (No safety) 0.5 (safety)*	**9800 (no safety) 1 (safety)**
Refutative	7	Traffic crash head injury	0.33	0.65	0.35	1.86	0.49	**1.4**

often are observed injuries real/not real?" The estimated pre-test probability is conditioned by the desired positive test result. This concept is explained in greater detail in the case analyses below:

4.2. Explanatory cases

(1) A traffic crash injury is described in which a minor severity (AIS 1) neck injury was deemed consistent with the injury potential of the collision.
   - Pre-test probability = 0.93. This value was estimated to be the proportion of litigants who have been diagnosed with a minor neck injury who have a real injury. The value was derived from a literature based assessment of rate of malingering and fraud (1 – [fraud and malingering rate] = rate of real injury). The highest estimate of malingering or fraud reported in the literature was 33% and the lowest was 7.5%[38–43] and thus the pre-test prevalence of real injury was estimated to range from 67–92.5%. The lowest rate of malingering was selected for the Error Odds assessment to favor the highest Error Odds for the test.
   - True positive rate = 0.5 (any injury), 0.5 (long term injury). The authors' reconstruction of the collision indicated a rearward speed change of 10.7 mph, and a Neck Injury Criterion (NIC) score of 25 m^2/s^2 was inferred from loads observed in the computer model. The injury potential of the collision was assigned a Factor of Risk of injury of 1.0 and 1.7 for long term and short term injury, respectively, based upon the NIC values reported by Boström et al. for 50 occupants with AIS 1 neck injuries from real world collisions (average 15 m^2/s^2), of which 11 lasted more than 6 months (average 25 m^2/s^2).[44] The injury rate at and above the neck injury threshold of 15 m^2/s^2 was 50% (25/50 injured above the threshold). The injury rate at and above the long term injury threshold was also 50% (6/11 injured). The small numbers in the sample increased the random error of the estimate, but this was not an issue as the conclusion from the data was *inclusive* rather than *exclusive* (see the previous discussion under Random Error).
   - False positive rate = 0.36 (any injury), 0.22 (long term injury). In the reference population described by Boström et al., at the injury threshold of 15 m^2/s^2 there were an estimated 44 out of 122 (36%) uninjured occupants and above the 25 m^2/s^2 threshold there were an estimated 27 of 122 (22%) uninjured occupants.

(2) A fall injury is described, which took place on a declining ramp. The claimant was a 61 year old female in whom Hayes et al. determined that a diagnosed fractured patella was more likely to have resulted from a forward-falling trip rather than a slip as claimed by the claimant, as the latter scenario would have been more likely to have produced a backwards or sideways fall.
   - Pre-test probability = 0.50. This value was estimated to be the proportion of litigants who report an inaccurate history of a fall either intentionally or mistakenly. There are no reference values from which to base this estimate, and thus the approximate range was derived from the values given previously for malingering and fraud (7.5–33%) plus an additional 50% to allow for error (12–50%). The highest rate was selected to favor a higher Error Odds calculation.
   - True positive rate = 0.89. As the basis for their conclusion that the fall was more probably the result of a trip rather than a slip Hayes et al. referred to study by Smeesters et al. of experimentally produced slips and trips that produced a 93–100% rate of forward falls associated with trips, compared with a 72–79% rate of sideways or backwards falls resulting from slips.[45] Absent the effects of bias this would equate to a true positive rate of 0.93–1, and a false positive rate of 0.21–0.29 (the complement of the rate of sideways or backwards falling from a slip was the rate of forward falling). An evaluation of whether the results of the study were extrapolable, without scaling, to the circumstances and individual at the center of the dispute demonstrates a cause for concern. Smeesters et al. evaluated the fall patterns of 14 athletic and fit men and women (seven of each), average ages 22.2 and 20.7, respectively, by using a level platform equipped with mechanisms to induce slips or falls. The study participants, who were equipped with knee pads, were instructed to not attempt any recovery during the fall on the platform, which was padded in all directions the fall could occur. The results of the study for slips and trips, as presented in Table 1 on p. 312 of the paper, indicate a forward fall rate of 93% for trips at slow gait speed and 100% at fast gait speed. In the same table, the forward fall rate for slips at slow gait speed is reported at 21% and 64% for fast gait speed (it is not apparent from the paper where the 21–29% used by Hayes et al. came from). The small subject numbers and dissimilarity of the conditions and participants in the study relative to the fall circumstances and characteristics of the litigant indicate the probable influence of both random error and bias in the extrapolation of the study results to the disputed matter as a an exclusionary boundary. To take this influence into account in the Error Odds calculation we decreased the true positive rate by a factor of (1.5 × [1 – true positive rate]) so that the adjusted rate was 0.86.
   - False positive rate = 0.64. The highest rate of false positives from the Smeesters et al. paper (the rate of slips that could produce a forward fall) was 0.64. To maintain the practice of underestimating, rather than overestimating the degree of error in the forensic biomechanical analysis this value was not adjusted, although it is as prone to random error and bias as is the true positive estimate.

(3) A fatal fall from a balcony is described. The decedent was a 57 year old man with a history of a seizure disorder in which it was determined that the cause of the fall over the balcony was the result of a seizure. Hayes et al. describe a computer model reconstruction of the kinematics of the fall, matched to the injuries sustained by the decedent. They describe their case question as "*whether or not the man could have fallen from the balcony as the result of a seizure*" (p. 76) but state their conclusions as "*the man fell from the balcony as the result of a seizure* (p. 77)". The shift from what was a *possible* or inclusive explanation for the cause of the fall to a *certain* or exclusive explanation is important from a forensic perspective, as the former is not admissible as evidence in most courts of law whereas the latter is.
   - Pre-test probability = 0.35. There is nothing in the biomechanical analysis of the fall that would indicate that the initiation of the fall was a seizure vs. any other cause. The fact that the decedent had a history of seizures only makes the explanation of a seizure as a cause of the fall possible, however the success of the medical management of the seizure disorder, the recent past history of seizure frequency, and the type of seizures that the decedent suffered from are all more valuable determinants that would condition the probability that the seizure was the cause of the fall.[46] Research concerning the epidemiology of fall-related injuries in patients with seizure disorders indicates that,

although they are prone to suffer fracture more than three times as often as the population without a seizure disorder, only 35% of the excess fractures in seizure patients are associated with a seizure event.[47]

- True positive rate = 0.35. As the biomechanical analysis is not a test for seizure disorder the true positive rate is the same as the pre-test probability.
- False positive rate = 0.65. This is the complement of the true positive rate.

(4) A fall injury on a trampoline is described. The fall resulted in a cervical spine fracture and spinal cord injury in a 40 year old man in whom it was determined that the forces generated at the neck if the feet slipped out from under the body while gently jumping on the trampoline were the most probable cause of the injuries, as opposed to the theory that he was attempting to perform a back flip. As with the prior case of the fall from the balcony, Hayes et al. initially state their goal as inclusive; "...*whether a slip and backward fall was a plausible mechanism for this injury* (p. 79)," but state their conclusions as exclusive; "...*the incident description provided by the subject was the likely mechanism for his injury and that he had not, as had been alleged, been attempting a back flip the first time he had set foot on a trampoline* (p. 80)."

- Pre-test probability = 0.53. While the forensic biomechanical analysis was used to establish that the force generated at the neck during a slip and fall were sufficient to generate supra-threshold AIS 3 and 5 forces in the neck using the N_{ij} compression values, this analysis could not be used to conclude that this injury mechanism was more probable than another mechanism (an attempted back flip) that could generate as much or more axial loading in the neck. As was the case with the previous seizure-fall case analysis, causal variables not addressed by the biomechanical analysis must be evaluated in order to properly condition the pre-test probability. The past behavior of the litigant (risky vs. conservative) is one consideration, and another is the epidemiology of trampoline-related injuries. In a study of 556 patients with trampoline-related injuries there were only eight cervical spine injuries, and only two of those were fractures.[48] For the entire study cohort 53% of the injuries were associated with an "awkward landing" on the trampoline, which would include a possible slip and fall mechanism.
- True positive rate = 0.53. As the forensic biomechanical analysis cannot additionally condition the probability of a slip and fall mechanism the true positive rate is the same as the pre-test probability.
- False positive rate = 0.38. Three of the eight cervical injuries occurred during an attempted somersault (the alternate scenario), a frequency of 37.5%.

4.3. Hypothetical cases

(5) A traffic crash fatality resulting from a high speed (74 mph) side impact collision and subsequent head injury is described. The risk of the observed critical severity (AIS 5+) head injuries is assessed for a belted and unbelted occupant. Hayes et al. conclude that the use of a seat belt would have likely eliminated the head injury but would have been likely to produce a critical neck injury instead.

- Pre-test probability = 0.8 (head injury), 0.53 (neck injury). Hypothetical scenarios require the estimation of both of the pre-test probabilities that are the source of the comparison. Hayes et al. do not provide a pre-test probability for the absence of a critical head injury in a belted occupant, leaving no means of assessing the accuracy of the conclusion that such an injury would have been *unlikely* had a belt been worn (in other words, given the magnitude of the collision, the injuries were likely regardless of restraint use). Thus for the Error Odds estimate the range of probability that a critical head injury would *not* have occurred in the restrained scenario was estimated to be low based on the high speed of the impact and the estimated HIC of 13,000 for the unbelted scenario, a value more than four times greater than the 99% risk threshold for critical head injuries as presented in Fig. 3 in the Hayes et al. paper. The result of this estimate was 0.5–0.8, a wide range meant to take into account the large degree of uncertainty in the estimate. The highest value was used for the Error Odds calculation. With regard to the neck injury risk, Hayes et al. reported the risk of critical neck injury as 53% for the calculated N_{ij} of approximately 3.0, and this was used for the pre-test probability for neck injury.
- True positive rate = 0.8 (head injury), 0.53 (neck injury). Although there is some potential for bias in the use of the N_{ij} for a side impact collision (it is derived from and validated with data on frontal collisions) the relatively large amount of data that it represents reduces the risk of random error and thus it was not adjusted. The basis for the true positive rate for the lack of critical head injury is stated above. Note that for this hypothetical scenario the pre-test probability is the same as the true positive rate.
- False positive rate = 0.2 (head injury), 0.47 (neck injury). These values are the complements of the true positive rates.

(6) The risk of a fatal head injury (an expanding subdural hematoma) is described for a 59 year old man who fell from an exercise bicycle onto a vinyl floor. The risk of the serious (AIS 3) severity injury was compared to the risk of the same severity of injury had the floor been padded, or if the man had been wearing a helmet or been seated on a recumbent (lower) bicycle. In their description of the case, Hayes et al. disclose that the decedent had been taking the anticoagulant medication Coumadin (generic name warfarin) prior to the fall. This case was the only one in which the authors did not reconstruct the forces of the fall. Instead they relied upon a fracture tolerance study of cadaver skulls in which it was determined that a HIC of 1000 was the threshold for fracture in the experimental population.[49] Hayes et al. noted that a subdural hematoma has an AIS severity level of three (serious) and that a HIC of 1000 corresponds to an AIS 3 or greater injury risk of 50–60%. The authors thus conclude that the HIC for the fall was at least 1000 because the presence of the subdural hematoma indicated that the HIC exceeded the risk threshold required to initiate a subdural hematoma (presumably 50%), but the HIC was not greater than 1000 because no skull fracture occurred. The authors did not provide any HIC estimates for the alternate scenarios, concluding only that floor padding would have prolonged head contact duration, a helmet would have attenuated impact force, and a recumbent bicycle would have reduced the fall height and resulting head impact force.

- Pre-test probability = 0.99 (no safety measures), 0.5 (safety measures). These values are the probability that the decedent would sustain an AIS 3+ injury without any safety measures in place, and the probability that

he would not have sustained an AIS 3+ injury if one of the safety measures been in place. The greatest complication to the analysis of the force required to cause the subdural hematoma seen in the decedent was his use of anticoagulant therapy, as this medication use is strongly associated with an increased risk of intracranial bleeding, and has been estimated to increase the risk of such bleeding by 7–10-fold.[50,51] Indeed, anticoagulated patients have been found to have sustained subdural hemorrhages following exposure to the relatively mild and typically non-injurious head accelerations that occur on roller coasters.[52] This leads to two difficulties with estimating a pre-test probability of injury for the forensic biomechanical analysis; first, the rationale employed by Hayes et al. in arriving at a HIC of 1000 (that a 50% risk of an AIS 3+ injury was the threshold for injury the decedent) cannot be extrapolated to an anticoagulated individual. As there is no lower boundary for the force required to cause the injury, then all that is left is the meaningless upper boundary of a HIC of 1000, below which a skull fracture would not be expected. What is known is that the fall and head impact did result in a subdural hematoma in the decedent, and thus whether the associated HIC was 1000 or 100, the pre-test probability for injury to this individual in this event is assigned a near certain probability of 0.99. The reduction of head impact force from an unknown amount would tend to reduce the risk of injury; however there is no way to know how much this would be, as the risk of injury may have been drastically reduced or not reduced at all by the safety measures. In order to complete the Error Odds analysis of this case study, however, the pre-test probability of head injury in the hypothetical scenario with safety measures is estimated to be half of the risk when no safety measures are present, or 0.50.

- True positive rate = 0.99 (no safety measures), 0.5 (safety measures). These rates are the same as the pre-test probabilities as the forensic biomechanical analysis could not additionally condition the probability of injury.
- False positive rate = 0.01 (no safety measures), 0.5 (safety measures). These values are the complements of the true positive rates.

4.4. Refutative case

(7) The authors describe a traffic crash case in which an unrestrained driver of a semi tractor-trailer was diagnosed with a concussion following a left frontal collision with another semi tractor-trailer. Hayes et al. conclude that the forces of the collision were insufficient to cause the concussion.

- Pre-test probability = 0.33. This value is the highest reported estimation of malingering and/or fraud in the population, and the complement of the pre-test probability used for the first case in the Explanatory category. It is likely that this value is significantly overestimated for two reasons; first, it is the highest value reported in the literature, and the majority of malingering estimates are substantially lower. Second, the rate was reported for all patients with a personal injury claim, rather than those who had claims that had progressed to litigation. It is reasonable to expect that some, if not many of the fraud and malingering cases would be identified prior to litigation.
- True positive rate = 0.65. Hayes et al. refer to the NFL football player study by Newman et al. as the source of their conclusion that a concussion was not consistent with the injury mechanism, as they indicate that the HIP_m they determined for the collision was 5.9 kW and that this corresponded with a concussion risk of less than 10% based on the logistic risk curve derived from the NFL football player data. The potential problems with bias and random error inherent with the use of these data have been discussed earlier in this paper. If the effect of random error (disregarding potential bias) is accommodated for by the bracketing of the data with a confidence interval then a HIP_m of 5.9 kW equates to a maximum concussion risk of approximately 0.35, rather than approximately 0.08. Thus the true positive rate is the probability of no injury at 5.9 kW, which is 0.65.
- False positive rate = 0.35. As discussed above, taking into account the random error in the Newman et al. data, the maximum probability that a concussion would occur at a HIP_m of 5.9 is approximately 0.35.

4.5. Limitations of Error Odds analysis

The Error Odds values for the various case studies as presented in Table 2 varied widely (from 0.3 to 9800), and depended primarily on the pre-test probability of the outcome of interest; the higher the pre-test probability the lower the error rate. Several points are worth noting when reviewing these results:

(1) A number of the input values were highly speculative and/or prone to bias and random variation, although when presented with a range of values on those which favored a higher Error Odds (and thus higher validity) value were used;
(2) Data that were not presented by Hayes et al. in their paper may have been present in the original analysis but not included in the case study, such as the HIC values for the hypothetical restrained traffic crash scenario in case 5. Had these data been used they may have altered the results of the Error Odds calculations;
(3) Although some of the input values were highly speculative, this was largely due to difficulties with the applicability of a biomechanical metric to circumstances in which the metric was inapplicable, and not to an inherent fault with the Error Odds methodology. Examples are the use of the HIC in an anticoagulated patient, or "biomechanical" measure of the probability that an epileptic patient fell during a seizure vs. some other cause, or whether a first time trampoline user was likely to have attempted a back flip. Intent, behavior, and pathology are not measured by biomechanical assessment.

Bearing in mind these caveats, the only case study in Forensic Biomechanics presented by Hayes et al. that had an Error Odds of greater than 10 was the first Explanatory case. None of the other case studies even exceeded an error ratio of 3 correct tests to 1 incorrect test. We find that the promise of Forensic Biomechanics as an ultimate test of truth in disputed matters must be tempered by the potential for error, depending upon how the technique is applied. In cases in which there is a less than 50% pre-test probability of a positive test result, for example when an injury that has been diagnosed and causally attributed to an injury mechanism is refuted by a biomechanical analysis that concludes that that the injury was improbable, the technique can be shown to be prone to the highest levels of error. On the other hand, when

the technique is used in instances with ≫50% pre-test probability it is more likely to surpass an Error Odds estimate of 10. We suggest that an Error Odds evaluation of forensic biomechanical analyses be incorporated with the methodology as a means of quality control.

5. Summary

The accuracy of Forensic Biomechanics largely depends on the circumstances in which it is applied and the pre-test probability of the condition of interest. The technique results in the most accurate results when used to explain how an injury occurred, vs. when it is used to refute the causal relationship between an injury mechanism and an injury. The results may be mixed when the technique is used to evaluate the probability of an injury outcome for an actual scenario vs. a hypothetical scenario; we suggest that the biomechanical analysis be correlated with observational epidemiologic study that supports the biomechanical conclusions. A large part of the difficulty with the practical application of Forensic Biomechanics stems from the dichotomization of continuous variables such as risk into an "injury likely" vs. "injury unlikely" delineation, as this raises the potential for false positive results, particularly when the technique is used in an exclusionary manner. An Error Odds analysis that takes into account the pre-test probability of the test result, as well as the true and false positive rate of the test is an important tool for evaluating the accuracy of the forensic biomechanical analysis. A forensic biomechanical opinion that is supported by an Error Odds estimate of 10 or more is more likely to survive a Daubert challenge in the courts.

Conflict of Interest

Both authors provide consultation in forensic matters.

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INFLUENCE OF CRASH SEVERITY ON VARIOUS WHIPLASH INJURY SYMPTOMS: A STUDY BASED ON REAL-LIFE REAR-END CRASHES WITH RECORDED CRASH PULSES

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ABSTRACT

Whiplash injuries resulting from rear impacts are one of the most important injury categories with regard to long-term consequences. Most rear impacts lead to no injury or to symptoms that are temporary. Impacts where the duration of symptoms differs need to be separated in analyses in order to isolate representative rear impact conditions in which more long-lasting whiplash injuries occur.

The aim of this study was to evaluate the influence of crash severity on symptoms duration of Whiplash Associated Disorders, WAD, separated for males and females, and for different grades of WAD (1-3) according to Quebec Task Force.

Since 1995, approximately 60 000 vehicles on the Swedish market have been equipped with crash pulse recorders measuring the acceleration time history in rear impacts. With the inclusion criteria of single rear-end crashes with a recorded crash pulse, and front seat occupants with no previous long-term AIS1 neck injury, 207 front-seat occupants in 150 crashes remained to be analyzed in this study, where the change of velocity and the crash pulse were measured.

A correlation was found between duration of symptoms and crash severity measured as mean acceleration and change of velocity. The risk of WAD symptoms for more than one month was found to be 20% at a change of velocity of approximately 8 km/h and at a mean acceleration approximately 5 g. A correlation was also found between grades of WAD and crash severity measured as mean acceleration and change of velocity. Out of all crashes with a recorded crash pulse only one out of 207 occupants sustained WAD symptoms for more than one month at mean acceleration below 3.0 g. Given the same crash severity, females had a higher risk of initial WAD symptoms than males.

INTRODUCTION

In the last decade some studies have been presented showing influence of duration of symptoms on crash severity in rear impacts. Regarding initial neck symptoms, the following studies describe the impact severity when no injury or short-term consequences occur. Hell and Langwieder (1998) found that most occupants sustained symptoms in impacts where the change of velocity was 10-15 km/h. Mc Connell et al (1995) performed low-speed rear impacts with seven male volunteers, with velocity changes of up to 10.9 km/h. None of the volunteers reported whiplash symptoms after a few days. Ono and Kaneoka (1997) and Siegmund et al (1997) found similar results from volunteer tests. In another study with volunteers (Eichberger et al 1996), where the sled impact velocities were 8-11 km/h and the mean deceleration 2.5g, the volunteers suffered whiplash symptoms for approximately 24 hours.

The influence of crash severity on more long-lasting symptoms is rarely studied. Based on a follow-up questionnaire with 65% answering frequency, Jakobsson (2004) found that 21% sustained long-term consequences in rear impacts with Volvo cars where the impact severity was defined as moderate. The impact severity “moderate” represented impacts in which the WHIPS recliner would have been activated. When the Volvo data was grouped according to whether the impact area involved rear members (reflecting a probable increase in the crash pulse amplitude) there was a tendency of higher initial AIS 1 neck injury risk for those with engaged rear members as compared to those with impact area outside rear members. Another study that tried to reflect the influence of the crash pulse on the injury outcome was Krafft (1998). It was found a relationship between the crash pulse on the neck injury risk in rear impacts, by showing that a longitudinally mounted engine (compared with a transversal one) in the striking car also increased the risk of long-term consequences in the struck car.

The influence of the crash characteristics on whiplash injury based on crash recording in real life rear impacts, has been presented earlier (see for example Krafft et al. 2002, and Kullgren et al. 2003). In these studies it was found that for the vast majority occupants that sustained symptoms for more than one month, mean acceleration was more than 4.5g and change of velocity higher than 10 km/h. Mean acceleration was found to be the best candidate to predict duration of symptoms compared to change of velocity and peak acceleration.

There is a need to further study the influence of crash pulse characteristics on AIS1 neck injury risks in rear impacts, both regarding kind of whiplash symptoms and duration of these symptoms. Furthermore, there is a need to separate the injury risk for gender. Several studies have shown that whiplash injuries occur more frequently among females than males (Berglund 2001, Maag et al 1993). However, there is always a problem with real-life data to handle the exposure problem concerning crash severity. With crash recorder data the outcome will be controlled for crash severity.

Based on more data from crash recorded rear impacts, the aim of this study was to evaluate:

- the influence of crash severity on the duration of symptoms of AIS1 neck injury in rear impacts.
- the influence of crash severity on whiplash symptoms classified according to Quebec Task Force.
- the influence of crash severity on the neck injury risk separated for males and females.

MATERIAL AND METHODS

Since 1995 crash recorders have been mounted under the driver or front passenger seat to document rear impacts in 60,000 vehicles in eight different car models of the same make. The models do not share the same seat type but are not separated in the analysis. All rear impacts since 1995 were reported to the insurance company Folksam, irrespective of repair cost. The inclusion criteria were single rear-end crashes with a recorded crash pulse, and front seat occupants with no previous long-term AIS1 neck injury. Out of 254 reported crashes, 150 crash pulses were recorded, in where 207 front seat occupants were involved. Out of these, 90 were men, 105 women, and in 12 cases the sex was unknown (10 were front seat passengers that were uninjured and 2 were drivers with initial symptoms but recovering within one month).

The remaining 104 rear impacts the trigger level of the CPR was not reached. In these crashes no acceleration pulse was measured, and they were not included in the analysis of this study.

Injury details were obtained from medical notes and interviews with the occupants. The interviewer had no information about the crash severity in each individual case. A follow-up of possible medical symptoms was carried out at least six months after the collision. The questionnaire of symptoms and the process of defining injury severity were structured in co-operation with a medical doctor. The symptoms noted were those associated with pain, stiffness and musculoskeletal signs, and with neurological symptoms, such as numbness. The duration of symptoms was defined as follow: no injury, symptoms less than one month, symptoms between one and six months, and for more than 6 months. The symptoms were also defined according to the Quebec Task Force on Whiplash associated Disorders (Spitzer et al. 1995).

WAD 0 – No complaints
WAD 1 – Neck complaints: pain, stiffness, or tenderness only
WAD 2 – Neck complaints and musculoskeletal signs
WAD 3 – Neck complaint and neurological signs

The Crash Pulse Recorder measures the acceleration time history in the principal direction of force during the time of impact. The crash pulses were filtered at approximately 100 Hz. The crash pulse recorder (CPR) has a trigger level of approximately 3g.

The development and accuracy of the CPR is described by Kullgren et al. (1995). Change of velocity and mean and peak accelerations were calculated from the crash pulse.

To visualize the influence of impact severity on risk of WAD, two kinds of plots were used. Injury risk versus impact severity was calculated for occupants with different duration of symptoms and for occupants classified in different grades of WAD. Injury risk was calculated as the proportion of injured occupants in each interval of impact severity. Intervals with less than 3 observations were excluded in the plots. In order not to force the injury risk curve into a specific shape, no mathematical function was used. The risk values for all intervals were connected using “smooth” curve fit in the software KaleidaGraph (Synergy software 2000).

In the second type, injury status in terms of duration of symptoms and grades of WAD, was, for all occupants, correlated with both change of velocity and mean acceleration in one plot.

RESULTS

Out of 207 front seat occupants in 150 rear impacts where the acceleration pulse was measured, 132 were uninjured, 75 reported initial symptoms whereof 51 recovered within a month, 7 sustained symptoms between one and six months and 17 had symptoms for more than six months after the impact. Out of the 207 occupants 49 were classified as WAD Grade 1, 20 as Grade 2 and 6 as Grade 3. In Table 1 the occupants are also divided according to seating position.

Table 1. Numbers of drivers and front seat passengers with different duration of symptoms and grades of WAD.

	Driver	FSP	Total
All	150	57	207
Uninjured	98	34	132
Symptoms < 1m	34	17	51
1m<symptoms<6m	7**	0	7
Symptoms > 6m	11*	6*	17
WAD grade 1	31	18	49
WAD grade 2	17	3	20
WAD grade 3	4	2	6

* One lumbar spine injury
** Two thoracic spine injuries

The occupants were also separated according to gender and seating positions. Table 2, 3 and 4 presents the number of male and female drivers, fronts seat passengers and front seat occupants together. It was found that the average impact severity was significantly higher for those occupants, both males and females, with symptoms for more than one month compared to the uninjured occupants.

Table 2. Numbers of male and female drivers and front seat passengers and average Δv and mean acceleration for different symptom durations.

	Males			Females		
	N	Δv	Mean acc.	N	Δv	Mean acc.
All	90	10.6	3.7	105	10.4	3.7
Uninj.	64	9.0	3.4	58	9.0	3.5
< 1 m	17	12.5	4.2	32	9.6	3.6
1-6 m	2	13.5	4.6	5	17.3	5.6
>6 m	7	19.9	5.2	10	17.6	5.1

Table 3. Numbers of male and female drivers and average Δv and mean acceleration for different symptom durations.

	Males			Females		
	N	Δv	Mean acc.	N	Δv	Mean acc.
All	75	10.3	3.7	73	10.2	3.6
Uninj.	55	9.2	3.4	43	8.9	3.3
< 1 m	13	12.3	4.2	19	8.9	3.5
1-6 m	2	13.5	4.6	5	17.3	5.6
>6 m	5	15.7	5.0	6	18.3	4.8

Table 4. Numbers of male and female front seat passengers and average Δv and mean acceleration for different symptom durations.

	Males			Females		
	N	Δv	Mean acc.	N	Δv	Mean acc.
All	15	12.3	3.8	32	10.9	4.0
Uninj.	9	7.8	3.2	15	9.4	3.9
< 1 m	4	13.2	4.2	13	10.8	3.7
1-6 m	0	-	-	0	-	-
>6 m	2	30.4	5.9	4	16.8	5.3

In the 104 rear impacts where the trigger level of the CPR was not reached and no crash pulse was recorded, one of the occupants had symptoms for more than six months, and one had symptoms between one and six months. None of the occupants was classified as WAD grade 3, but two as WAD grade 2. All other occupants were either uninjured or reported initial symptoms, but recovered within a month. As the trigger level of the CPR is approximately 3 g, the mean acceleration must in these crashes be below 3 g.

The numbers of occupants with different duration of symptoms and those classified in different grades of WAD, is presented in intervals of impact severity in Figures 1 to 4. The information in these figures is used to calculate the injury risk in each interval of impact severity, presented in Figures 5 to 8. In the interval 5-10 km/h 7 occupants had symptoms for more than 1 month. Out of these occupants 6 had a mean acceleration above 3.3 g, and all had a mean acceleration above 2.8g, see Figure 9. From the information in Figures, 1, 2 and 9 it appears like mean acceleration to a higher extent than change of velocity influences risk of WAD.

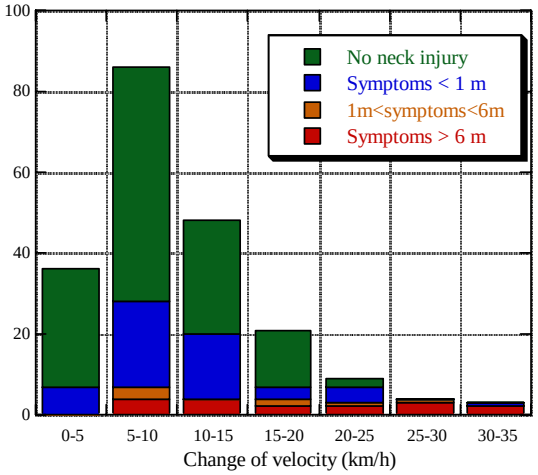

Figure 1. Numbers of injured and uninjured occupants in intervals of change of velocity.

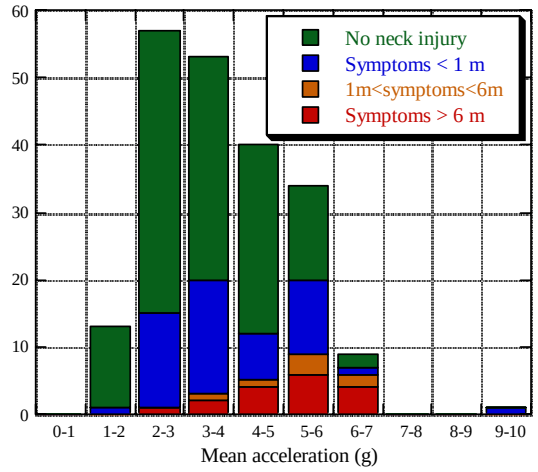

Figure 2. Numbers of injured and uninjured occupants in intervals of mean acceleration.

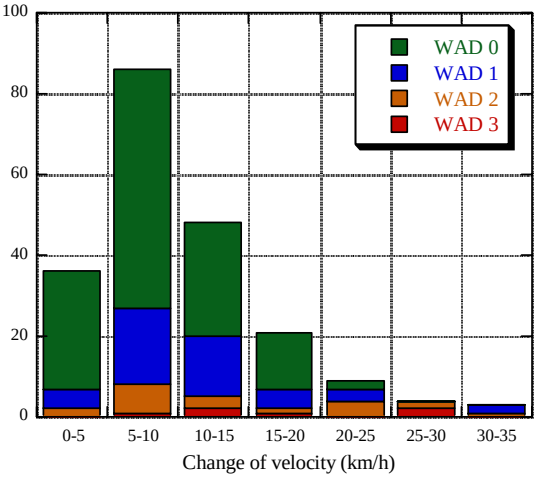

Figure 3. Numbers of injured and uninjured occupants in intervals of change of velocity.

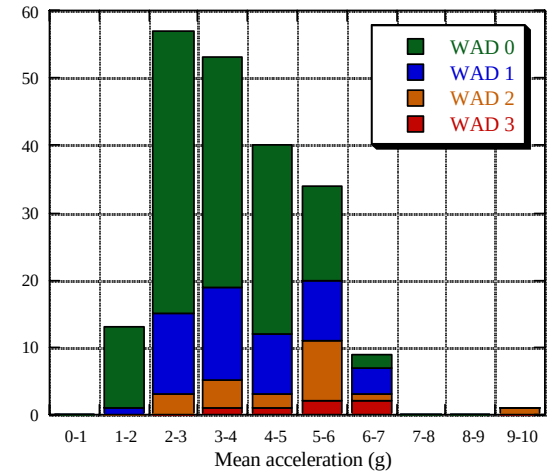

Figure 4. Numbers of injured and uninjured occupants in intervals of mean acceleration.

A correlation between injury risk and change of velocity was found for initial and more long lasting symptoms, see Figure 5. At a change of velocity above 20 km/h the risk of long lasting symptoms increase with a high rate. Risk of symptoms for more than one month was found to be 20% at approximately 18 km/h.

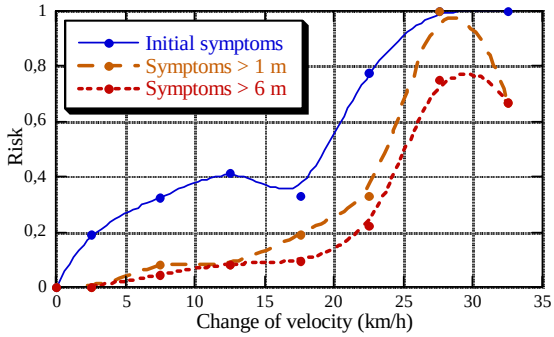

Figure 5. Injury risk in intervals of change of velocity for occupants with initial and long-term symptoms.

Injury risk and mean acceleration was also found to be correlated, see Figure 6. The risk of symptoms for more than one month was 20% at a mean acceleration of 5 g. Above 5 g the risk increases with a higher rate than below 5 g. In Figure 6 the occupant with initial symptoms at a mean acceleration of 9.1 g is not included.

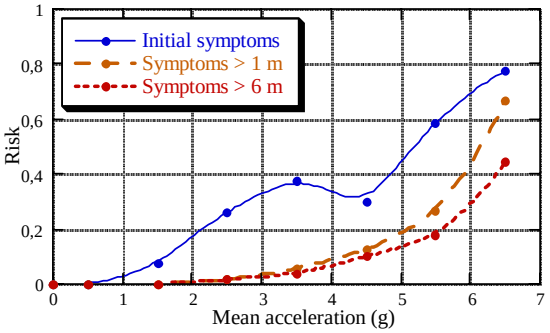

Figure 6. Injury risk in intervals of mean acceleration for occupants with initial and long-term symptoms.

Increased impact severity, both in terms of change of velocity and mean acceleration, was found to increase the risk of WAD symptoms, see figures 7 and 8. Furthermore, the risk of neurological symptoms of WAD, grade 3, was found to be lower than for grade 1 and 2 for the whole range of both change of velocity and mean acceleration. The risk of symptoms of grade 2 appears to increase above 17 km/h or 4.5 g.

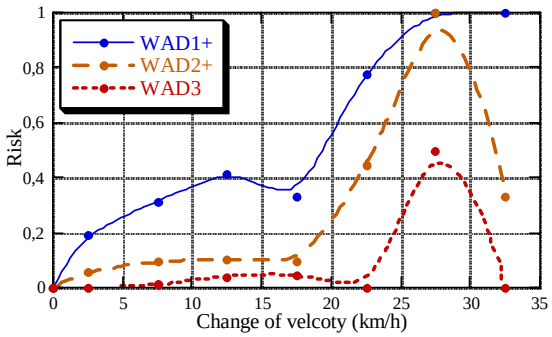

Figure 7. Injury risk in intervals of change of velocity for occupants classified as different grades of WAD.

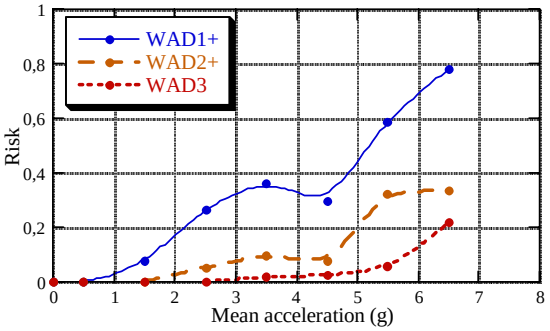

Figure 8. Injury risk in intervals of mean acceleration for occupants classified as different grades of WAD.

Only one of the 24 occupants with symptoms more than one month had a mean acceleration below 3 g (2.8 g). All other occupants with symptoms for more than one month had a mean acceleration above 3.3 g, see Figure 7.

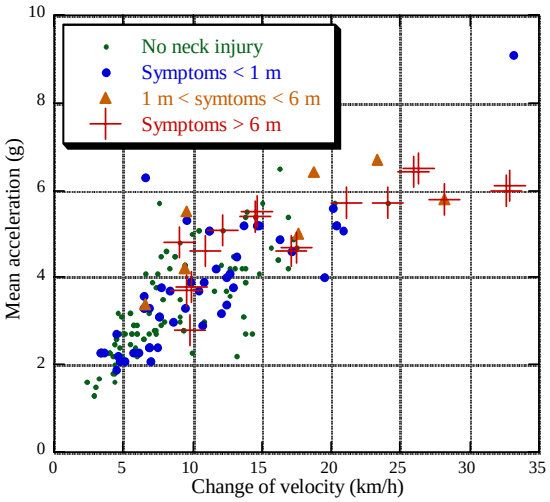

Figure 9. Change of velocity versus mean acceleration for occupants with different duration of symptoms.

Most occupants defined as WAD grade 3 had a mean acceleration above 4 g, see Figure 10. Occupants with a WAD defined as grade 2 seem to occur in a wide range of both change of velocity and mean acceleration.

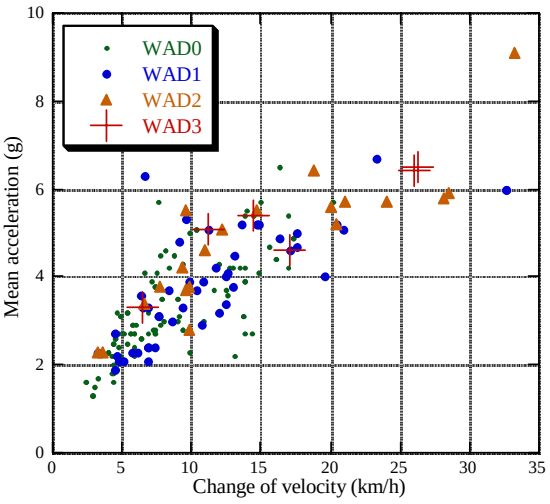

Figure 10. Change of velocity versus mean acceleration for occupants classified in various grades of WAD.

The risk of initial symptoms was found to be higher for females than males, both regarding change of velocity and mean acceleration, see Figures 11 and 12. Females appear to more often sustain initial symptoms at lower impact severity than males, especially regarding mean acceleration.

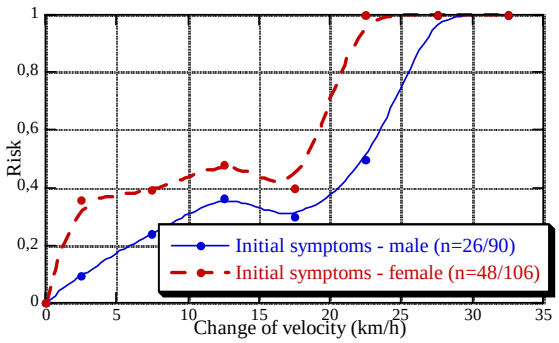

Figure 11. Risk of initial symptoms in intervals of change of velocity for males and females.

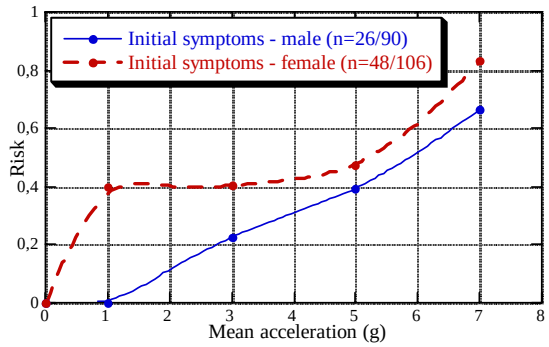

Figure 12. Risk of initial symptoms in intervals of mean acceleration for males and females.

DISCUSSION

Knowledge about the correlation between crash severity and injury risk is essential to more effectively prevent injuries in car crashes. The data used in this study mean a unique opportunity to analyze how acceleration influences the risk of whiplash injury. In a previous study (Krafft et al 2002) the crash pulses from 66 rear impacts have been presented, but in this study the data is more comprehensive and therefore more reliable conclusions can be drawn. The combination of valid and reliable impact severity measurements and prognostic injury data made it possible to study relations that would otherwise be difficult to obtain.

It is not possible to objectively determine the diagnosis of WAD, therefore the credibility of these injuries is often raised. In this study the injury data were mainly obtained by interviews with the occupants, which might influence the outcome. Better significance could be expected if only symptoms verified by a medical doctor were used. However, to minimize the risk of biased data, the interviewer had no knowledge about the crash severity in each case.

The results are based on seven different models from one car manufacturer. The limits in crash severity for different injury levels may therefore be different for other vehicles.

In all figures, the results were based on the rear impacts where fully crash pulses were recorded. This fact influences the results where the correlation between crash severity and injury outcome was analyzed. The results in Figure x-xx show no difference in crash severity for the occupants that sustained no injury and those with symptoms for less than one month. However, the impacts where no crash pulse could be recorded were not included in the study. A difference in crash severity could therefore be expected between the uninjured occupants and those with short-term consequences.

A correlation between crash severity and duration of symptoms was found. Other studies (Jakobsson 2004, Olsson et al 1990) did not find a relationship between impact speed (EBS) and the initial spectrum of symptoms or duration of symptoms. However, EBS or change of velocity calculated with retrospective methods has too low accuracy to predict the crash severity (Kullgren 1998), especially in low speed impacts (Lenard et al. 1998).

When designing test methods for evaluating vehicle crashworthiness with regard to whiplash, the results show that the acceleration pulse differs considerably, depending on whether the focus is on short- or long-term consequences. If too low crash severity is chosen, there is a risk of sub-optimization against short-term consequences. To create conditions for a robust anti-whiplash system it is advisable to have at least two tests at different crash severity levels: one test representing the crash severity where the risk of long-term consequences is high, and another one representing a lower limit above which most of the whiplash injuries occur (symptoms more than one month).

The severity of the initial neck injury was classified according to the Quebec Task Force injury scale WAD 1-3. The duration of symptoms appears to better correlate with crash severity than WAD. This is logical since the WAD-scale is supposed to predict long-term injury outcome. Using the WAD-scale is a round-about way of describing the duration of symptoms, but less reliable. At least when the WAD classification was based on interviews with the occupants. The quality

of the classification would probably be better if it was based on medical examination of the occupants. In this study, the WAD 2 were found at all crash severity levels, but WAD 0 and 1 predominated in the lower severity segment. Whiplash injuries with neurological signs, WAD 3, occurred mostly at higher mean accelerations (above 4.5g), but they represented only six occupants.

Given the same crash severity level, females were found to have a higher risk of initial symptoms. If focusing long-lasting symptoms there is a need of more data to separate risk curves for males and females. Most studies, controlled for position, show a higher injury risk (long-term) for females (Jakobsson 2004, Krafft 1998) than males but there is no control for the exposed crash severity in the impacts. However, given the same crash severity there is a high probability that females still have a higher risk. It is important for preventative measures to determine critical crash severity levels mainly based on data related to females, ant not based on mean values for the total population.

CONCLUSIONS

- A correlation was found between duration of symptoms and crash severity measured as mean acceleration and change of velocity. The risk of WAD symptoms for more than one month was found to be 20% at a change of velocity of approximately 8 km/h and at a mean acceleration approximately 5 g.
- A correlation was found between grades of WAD according to Quebec Task Force and crash severity measured as mean acceleration and change of velocity.
- Out of all crashes with a recorded crash pulse only one out of 207 occupants sustained WAD symptoms for more than one month at mean acceleration below 3.0 g.
- Given the same crash severity, females had a higher risk of initial WAD symptoms than males.

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